A guide to passing the fellowship examination in general surgery

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Preface

Our life is what our thoughts make it.
(Marcus Aurelius Antonius, 121–180).

The fellowship examination in general surgery has a reputation for being one of the most difficult and rigorous postgraduate examinations. General surgery is, by necessity, a discipline that covers a broad range of surgical conditions incorporating a large number of topics. Consequently, trainees are expected to have acquired a tremendous amount of technical and non-technical knowledge at completion of training. Accordingly, the breadth and depth of basic and clinical science, clinical practice and operative surgical knowledge assessed in the fellowship examination is formidable. It is not surprising, therefore, that success rates in the examination consistently hover around the 50–60% mark. For most candidates, this examination is the most important of their life and represents the final barrier to independent practice as a specialist general surgeon, frequently reflecting the culmination of 25 to 35 years of education since childhood.

Examination surgery represents the synthesis of our collective experience of delivering tutorials, lectures and ‘mock examinations’ for over a decade to candidates preparing for the fellowship examination in general surgery. Whilst there can be no substitute in the examination for knowledge gained by hard work, clinical experience and appreciation of the published literature, the fellowship examination is designed to test clinical wisdom, judgment, insight and safe practice and not just encyclopaedic knowledge. Accordingly, the aim of this book is to use a practical approach to provide candidates with appropriate direction, focus and instruction on how to organise their studies and to prepare for each component of the examination with particular emphasis on examination technique to optimise performance. It is most certainly not intended to replace other reference textbooks for the various components of the examination.

One of the most significant challenges of embarking on a project such as this is to produce a book that is relevant and accurately reflects the material encountered in the examination. To this end, we have collected and catalogued past papers and questions from the last 10 years to assemble a list of the most frequently encountered topics. This information has been distilled into the respective sections of this book that mirror each of the individual components of the current fellowship examination, namely: (i) the written papers; (ii) the clinical examinations; and (iii) the viva voce examinations. Within each of the sections of the book, we will attempt to simplify and demystify the common questions and scenarios frequently encountered in the examination and present a structured approach, incorporating suitable techniques and strategies that we have developed and refined from our own experiences of preparing for examinations and from being involved with the education of postgraduate trainees in general surgery sitting the fellowship examination. Using sample ‘model answers’, the purpose of this approach is to equip candidates with the confidence to address common cases/topics, but it will also enable individual candidates to develop a reliable, systematic approach to successfully address any scenario that they may encounter in the examination.
It must be emphasised that we are most certainly not advocating that the model answers contained within this book are the only way to tackle examination questions. Indeed, you personally may have a different style or technique and you will certainly hear some of our recommendations challenged by your mentors. This epitomises surgical practice, where different general surgeons manage different conditions or perform different operations in different ways. *We are merely presenting one way of answering the questions that we, and our numerous students over the years, have found useful and successful in the fellowship examination.* We urge you to refrain from getting bogged down in the debate of which approach is right or wrong, but instead concentrate on understanding the arguments surrounding many of these key issues and incorporate different techniques and advice from your peers and mentors, as well as from this book, into your own answers.

While this book is aimed squarely at candidates preparing for the fellowship examination in general surgery of the Royal Australasian College of Surgeons, its contents will also be relevant to candidates preparing for the fellowship examination of the Royal Colleges of Surgeons in the UK and Ireland (Intercollegiate Specialty Examination), Canada, South Africa and Hong Kong, as well as having general application to all general surgeons, surgical residents and medical students.

Today's trainees are dependent on the enthusiasm and commitment of their trainers, supervisors and surgical mentors to mould them into tomorrow's surgeons. We sincerely hope that this book furnishes candidates with the appropriate tools to successfully negotiate the examination as they progress to becoming the future generation of specialist surgeons.

Good luck!

Christopher J Young
Marc A Gladman
Chapter 3.3

Common medium cases

3.3.1 Breast cancer case

Figure 3.3.1A
Mammogram with possible microcalcification in left breast, MLO views on left, CC views on right

Figure 3.3.1B
Ultrasound of solid breast mass
One in nine Australian women will develop breast cancer before the age of 85 years, so breast cancer cases are common. Typical cases include women who have had recent surgery or who are in the middle or have just completed multidisciplinary complex treatment. You may well get a complete structured history from a single open question. Allow time for the patient to talk and it will become obvious whether or not this is the case. If it seems that much information is missing, then some prompting will be required. The patients are usually surprisingly well-informed and will often use jargon accurately such as sentinel node or DCIS.

You may be introduced to a woman sitting in a clinic room as follows: ‘This is Mrs Jones, would you please take a history from her and perform an appropriate examination’.

**The history**

- Introduce yourself and ask the patient what has been going on. The patient may indicate that she has had a breast cancer removed 4 months ago.
- Try to maintain a structure to your history, using the framework above. After orientation, ask questions specific to the breast problem.
- The presenting symptoms and history of presenting complaint:
  - *presentation*: presenting clinical symptoms (presence of a lump, pain, breast skin change, nipple change or discharge), or asymptomatic detection during regular check-up or screening mammogram
  - *investigations*: radiology/biopsy
  - *treatment*:
    - Was neo-adjuvant treatment required or discussed?
    - What surgery did she have done? Establish whether the initial operation was mastectomy or lumpectomy and method of axillary staging. Has a re-operation been performed (cavity re-excision or mastectomy or axillary lymph node clearance). If so, ask why re-operation was required (positive margin or positive sentinel node).
    - Did she undergo any breast reconstruction?
    - Were there any complications (seroma, shoulder pain or lymphoedema)?
    - What adjuvant treatment was required (radiotherapy or chemotherapy)? What chemotherapy? Is she taking tamoxifen or other hormonal therapy?
    - What follow-up imaging has she had?
- Risk factor assessment:
  - oestrogen and reproductive history: age at menarche, menopause, number of children, contraceptive and hormone replacement therapy.
- Past medical history.
- Surgical history including previous breast lumps or breast cancer.
- Drug history and allergies.
- Family history — particularly of breast or ovarian cancer.
- Social history: occupation, home circumstances, smoking tobacco and alcohol consumption.

**The examination**

- Preparation — (see above):
  - position: edge of the bed
  - remove upper garments including brassiere.
Breast examination:
- inspection:
  - breast asymmetry/skin changes, including nipples and areolar
  - scars from the previous surgery breast/axilla
  - tattoos from radiotherapy (usually on sternum and lateral chest wall)
  - evidence of chemotherapy port on the chest wall
  - the back of the chest for a scar from a latissimus dorsi flap
  - enhance inspection with movement: ask the woman to put her hands on her hips and squeeze her hips together
  - ask her to raise her hands above her head and look for tethering of the skin.
Position change: ask the woman to lie down on slightly reclined couch (if available).
- Palpation:
  - each breast and ipsilateral axilla, including nipples and areolar
  - the supraclavicular fossa bilaterally
  - with the patient still recumbent, examine the abdomen briefly by inspection (looking for scars from TRAM flap) then palpation of the liver edge.
- Auscultation:
  - sit the patient forward and auscultate to the lung bases.
- Conclusion:
  - thank the patient for taking the time to come to help with your exam
  - help her off the couch if required and to find her clothing if necessary
  - wash or sanitise your hands again at the end of the examination if possible.

The examiners will then commence discussion in the same room or another room, depending on the venue.

The discussion
Breast cancer diagnosis is best achieved using triple assessment:
- clinical
- imaging: mammogram and ultrasound
- pathological: FNAC or core biopsy.

INVESTIGATIONS
- Imaging — mammography.
  - The standard for mammography is a bilateral mediolateral oblique (MLO) and cranio-caudal (CC) view.
  - These are low energy x-rays (30 kVp) obtained by parallel plate compression to even the thickness of the breast tissue.
  - The images are assessed for symmetry, architectural distortion, masses, calcifications and densities.
  - Fig 3.3.1A shows a bilateral mammogram with MLO and CC views. The MLO is shown on the left and is distinguished by the pectoral muscle shadow. The convention is to arrange them on the viewing box as shown.
  - For any mass describe the shape, margin, density and associated microcalcifications. Round or oval shapes are typically benign (fibroadenoma or cyst). Lobulated or irregular masses are typically malignant. Margins that are circumscribed are benign, whereas microlobulated, obscured, indistinct or spiculated outlines are suspicious for malignancy. For calcifications describe the shape, location, number and distribution. There are a variety of typically benign calcifications such as skin, vascular, popcorn-like, rod-like, round, punctuate, milk of calcium, eggshell, dystrophic and suture. Of intermediate concern are amorphous or indistinct
calcifications. Microcalcifications that are pleomorphic or heterogeneous, fine/linear and fine or linear/branching calcifications have a high probability of malignancy.

- **Imaging — ultrasound.**
  - Breast ultrasound uses higher frequency sound waves (typically 8–13MHz) than used for abdominal ultrasound, offering greater resolution but less penetration.
  - Benign features include ellipsoid shape, hyperechogenicity or anechoic, and smooth, well-circumscribed margins. Cysts are oval or round, well circumscribed, anechoic with posterior acoustic *enhancement*. Features of malignancy are irregular margins, hypoechoic to the surrounding tissue, with posterior acoustical *shadowing*. Malignant masses are usually taller than they are wide.
  - Fig 3.3.1B shows an ultrasound of a breast mass demonstrating many of the features of malignancy.

- **Pathological: FNAC.**
  - FNA uses a 22G needle and a syringe (aspirated to include several ml of air), which is passed into the mass if clinically apparent or using ultrasound guidance.
  - It has a false positive rate <1%, a false negative rate <10%. Insufficient material for diagnosis occurs in about 10% of cases.
  - The presence of carcinoma cells does not differentiate between invasive and in situ breast cancer.
  - FNA results are reported as:
    - C1 — inadequate/acellular
    - C2 — benign
    - C3 — atypical
    - C4 — suspicious or
    - C5 — malignant.

- **Pathological: core biopsy.**
  - Conventional core biopsy uses a spring-loaded biopsy gun unseeing a double-action needle (typically 14G) consisting of an inner trocar with a sample notch and an outer cutting cannula.
  - To obtain the specimen the needle must be withdrawn. Usually, 4–6 samples are taken (4–6 insertions).
  - Core biopsy provides a diagnosis of histological type, grade, hormone receptor status, HER2 expression.
  - Core biopsy differentiates DCIS from invasive cancer.

**TREATMENT**

- Treatment should be discussed at an MDT with medical and radiation oncologists involving review of the pertinent radiology and pathology.
- It should be considered with three dimensions: (1) treatment of the breast; (2) staging and treatment of the axilla; and (3) systemic treatment.

**Treatment of the breast**

- Options are mastectomy (with/without immediate or delayed reconstruction) or wide local excision (WLE) to clear margins combined with post-operative radiotherapy (XRT).
- The risk of local recurrence with WLE alone is 30–40%. The risk of local recurrence may be higher with breast conservation and XRT than mastectomy but the long-term survival is the same.
- Breast conservation should be considered if a satisfactory cosmetic result can be achieved (which is less likely when >10–20% of breast volume is removed).
If the woman has a large breast, especially if upper/outer quadrant, WLE is often a good option. For a woman with a smaller breast cosmetic outcome may be unsatisfactory. Oncoplastic techniques can be used to facilitate reconstruction of the conserved breast (possibly combined with contralateral balancing procedure).

**Staging and treatment of the axilla**

- LN status is the strongest prognostic determinant in early breast cancer.
- If the axilla is clinically positive with palpable nodes, then a core biopsy or FNA should be performed. If cancer is confirmed then axillary lymph node dissection (ALND), generally to level III, will be required.
- If the axilla is clinically and radiologically negative then a staging procedure is required. Options are: sentinel lymph node biopsy (SLNB) and axillary lymph node dissection.
- SLNB is performed using radio labelled colloid and/or blue dye. SLNB has a <10% false negative rate in experienced hands. This is an acceptable staging procedure for the axilla. If positive then ALND is performed either at the time (if frozen section is used) or after definitive pathology is available. In 80% of cases where SLNB is positive for malignancy, the SLN is the only positive node found in the axilla.
- The benefit of completion ALND is currently the subject of RCTs. There are four large RCT of SLNB (NSABP B-32, Almanac, SNAC and Milan study). These have enrolled patients with clinically negative axilla and cancer less than 3 cm. The follow-up of these studies is currently relatively short and most demonstrate only the false negative rate (negative SLNB where subsequent ALND showed lymph node metastasis), usually of 5–15%. The oncological implications of the false negative rate are currently unknown. Most studies have identified reduced short-term morbidity (pain, shoulder stiffness and lymphoedema) with SLNB.
- Traditionally, ALND to level II or even level I (node sampling) has been performed in the clinically negative axilla for staging. This has been replaced by SLNB in many centres but remains a treatment of proven efficacy where skills or equipment for SLNB are not available.
- ALND should be omitted in cases of DCIS as rates of metastases is <1%.

**Systemic treatment**

- Adjuvant systemic treatment involves the use of cytotoxic or endocrine therapy after local treatment of breast cancer to eliminate clinically occult micrometastases, to prevent local recurrence and improve survival.
- Adjuvant endocrine therapy reduces local recurrence and contralateral breast cancer development by 50% and mortality by 25%.
  - Tamoxifen is generally offered if the cancer is ER/PR positive and should be taken for 5 years. Its use is associated with increased risk of endometrial cancer (excess mortality 1–2/1000), DVT/PE, stroke, flushing, vaginal dryness, weight gain, nausea, disturbances hair and nail growth and reduced metabolism of warfarin.
  - GnRH agonists (zoladex) overstimulate and subsequently down-regulate GnRH receptors to achieve postmenopausal levels of estradiol and can be used in pre-menopausal women.
  - Aromatase inhibitor (such as anastrozole) may be used in post-menopausal women upfront (better than tamoxifen), switching from tamoxifen (better than tamoxifen) or after tamoxifen (better than placebo). They increase GnRH in pre-menopausal women with the undesired effect of increasing.
3.3 Common medium cases

- Chemotherapy produces a relative risk reduction (RRR) of recurrence of 20–35%, and of death by 10–30%. The same proportional RRR is seen in all subtypes of breast cancer. However, the absolute benefit depends on the risk of recurrence or death for that patient with those with the highest chances of recurrence receiving greatest absolute benefit.
- Women under 70 years with node-positive disease should be offered adjuvant chemotherapy, but the benefit is greatest in women under 50 years.
- Taxanes are preferred agents.
- In patients with lower risk of recurrence and death the absolute benefit is lower and the risks and side effects of chemotherapy may exceed its benefits. However, there is a group of patient with node-negative disease who may benefit from adjuvant chemotherapy. These include: (a) tumour > 2 cm; (b) Grade II/III; (c) Lymphovascular or perivascular invasion present; (d) age <35; (e) ER/PR negative; and (f) HER-2 +ve.
- Signal transduction inhibitors:
  - trastuzumab (Herceptin) for at least 1 year in HER-2 positive disease
  - achieves a 50% reduction in risk of recurrence and 33% reduction in risk of death.
- Metastatic systemic therapy:
  - bisphosphonates decrease skeletal events in metastatic disease but do not reduce rate of bone metastases in early breast cancer
  - signal transduction inhibitors (trastuzumab — Herceptin) are effective in HER-2 positive tumours and is usually given with chemotherapy.

Extra notes

INVASIVE CANCER

Ductal (no special type) carcinomas account for 70–80% of breast cancers, lobular carcinoma account for 20% and the remaining 10% are mucinous, tubular or medullary.

IN SITU CANCER

- DCIS is a pre-invasive, in situ lesion of the breast where malignant epithelial cells are found confined within the basement membrane. The architectural types of DCIS are comedo, solid, cribriform and papillary/micropapillary.
- When a few cells penetrate the BM with no focus of invasion >1 mm, the disease is deemed ‘microinvasive’.
- DCIS represents 5% of all symptomatic breast cancers, but 20% of screened breast cancers.
- About two-thirds of invasive breast cancers have associated DCIS. The risk of invasive breast cancer in patients with DCIS is 10x the normal population, the majority in the ipsilateral breast.
- On mammography, DCIS tends to be present as clustered, pleomorphic and branching or linear calcifications. Given that FNA cannot differentiate invasive carcinoma from DCIS, a tissue biopsy should be used for treatment planning of suspected DCIS.
- The vast majority of DCIS is not palpable, so tissue diagnosis must be obtained using hookwire localisation or stereotactic core biopsy (when a carbon tack is placed to mark the site).
- Small, localised areas of DCIS (<4 cm) should be treated by breast-conserving surgery with or without radiotherapy. Impalpable lesions require radiological localisation pre-operatively.
- The NSABP B-17 and EORTC 10853 studies showed that RXT reduces the risk of local recurrence of DCIS by about 50% compared with excision alone and reduced
the proportion of recurrence with invasive pathology. The Van Nuys prognostic index (VNPI) is a scoring system, which generates a score based on these factors to help guide appropriate treatment based on the risk of recurrence, but has not been validated in a prospective trial.

- Mastectomy is indicated for widespread contiguous or multi-focal DCIS, where adequate excision cannot be achieved with a cosmetically acceptable wide excision. Widespread microcalcification (on pre-operative mammogram) in the presence of proven DCIS, or persistent positive margins when attempting to excise DCIS are also indications for mastectomy.
- Positive axillary lymph nodes are seen in <1% and thus axillary staging can be avoided.
- LCIS is a monomorphic proliferation of cells in one or more terminal ducts or acini.
- It is uncommon and usually found incidentally at histology (about 1% of breast biopsies) and is usually found in perimenopausal women (median age 40–45 yrs), and has a decreasing incidence after menopause.
- It is multicentric and/or multifocal and/or bilateral in up to 60%. It is considered to be an index of risk for invasive cancer in either breast, rather than a premalignant lesion, with a 25 to 30% long-term cancer risk over 20 years.
- If LCIS is picked up on biopsy, excision and clear margins are probably unnecessary, as the abnormality cannot be imaged, and there is multicentricity and bilaterality. Need to consider excision to clear margins in cases of pleomorphic LCIS (histologically aggressive variant), especially in young patients. Follow-up involves lifelong surveillance, including annual mammography (at least for 10 yrs until age 50) and examination plus axillary clinical review.

For breast cancer please also see 2.2.2, 2.2.4, 4.2.2, 4.4.3, and 4.6.1.

### 3.3.2. Rectal cancer

![Rectal cancer colonoscope image](image)

**Figure 3.3.2A**
Rectal cancer colonoscope image
One in 12 Australians will be affected by large bowel cancer by the age of 85 years, so colorectal cancer cases are common.

You may be introduced to a man sitting in a clinic room as follows: ‘This is Mr Jones. Would you like to take a history from him and examine him as is appropriate’.

The history

- Introduce yourself and ask the patient what has been going on. The patient may indicate that he had an operation for rectal cancer last week and now has a stoma bag for 3–6 months.
- Try to maintain a structure to your history, using the framework above. Begin by orientating yourself.
- The presenting symptoms and history of presenting complaint:
  - presentation — bleeding per rectum (PR) and its detail; altered bowel habit (constipation/ diarrhoea/both); tenesmus; mucous discharge per rectum; abdominal pain (SOCRATES) or distension
  - associated symptoms of the diseased (GI) system: nausea; vomiting; jaundice
  - other associated symptoms — systemic: weight loss; anorexia; malaise; shortness of breathe
  - other associated symptoms — pelvic: pneumaturia; UTI; haematuria
  - investigations: colonoscopy/staging radiology (CT/MRI)/biopsy.
- treatment:
  - Was the patient admitted electively or acutely?
  - What procedure was performed?
  - Was neo-adjuvant treatment required or discussed?
  - Were there any complications after the operation?
  - Current status of the patient (diet, mobility, bowel function, expected date of discharge).
  - Will adjuvant treatment be required (radiotherapy or chemotherapy)?
• Risk factor assessment:
  • personal or family history of polyps/colorectal cancer/inflammatory bowel disease.
• Past medical history.
• Surgical history.
• Drug history and allergies.
• Family history — particularly of endometrial/gastric/ovary/urothelium (HNPCC).
• Social history: occupation, home circumstances, smoking tobacco and alcohol consumption.

**The examination**
• Preparation — see above:
  • position the patient flat with one pillow under the head
  • expose the abdomen from nipples to pubic symphysis but state that ideally you would like to expose the patient to mid-thigh.
• General examination from the end of the bed:
  • overall condition
  • drains, tubes, infusions, TPN, IDC, oxygen etc.
• Assessment for peripheral signs, unless instructed otherwise:
  • vital signs
  • hands: stigmata of gastrointestinal disease — anaemia/clubbing/malnutrition
  • head and neck: cachexia (wasting of temporalis)/jaundice/pallor/lymphadenopathy.
• Examination of the abdomen:
  • inspection: moving with respiration; distension; scars; drains; stomas; dressings; bruising. Inspect wounds for signs of infection. Look at drains, stomas and urine bag if present
  • palpation: ask if there is any pain. Soft; tender; organomegaly; ascites. Assess groin and incision for evidence of a cough impulse. Palpate the testicles
  • percussion: not usually relevant post-operatively unless distended and you want to determine if it is gaseous or fluid (ascites/ileus). Percussion for shifting dullness; fluid thrill
  • auscultation: listen in four quadrants for the character of borborygmi, bruits over the renal arteries, aorta and liver.
• Conclusion:
  • say that you would do a digital rectal examination in the pre-operative setting (distance of cancer from the anal verge; prostate; sphincter tone)
  • cover the patient
  • reposition the patient
  • thank the patient
  • clean your hands again
  • only now turn to face the examiners.

**The discussion**
• The examiners may ask you to tell them what you found. An example of a succinct summary for this case would be as follows, ‘Mr Jones is a 64-year-old man who had a rectal cancer found at colonoscopy after presenting with 3 months of rectal bleeding. He was found to have a low rectal cancer and after CT scan of his abdomen and pelvis and a MRI he proceeded to have pre-operative radiotherapy for 6 weeks followed by an ultra-low anterior resection and a diverting loop ileostomy one week ago.’
3.3 Common medium cases

- Investigation of a patient with rectal cancer
  - Blood tests:
    - FBC, EUC, LFTs, CEA.
  - Colonoscopy:
    - to exclude synchronous colonic neoplasms.
  - CT scan chest, abdomen and pelvis — staging to detect metastatic disease.
  - TRUS/MRI pelvis and PET scans where indicted.
    - TRUS/MRI determine the depth of invasion through the bowel wall (T staging) and spread to adjacent nodes to help decide which patients need neo-adjuvant therapy.
  - Examination under anaesthetic:
    - tumour location (anterior/posterior/circumferential), height (from anal verge), relationship to the anal sphincters and depth of penetration (T stage) can be estimated. Superficially invasive tumours are mobile, whereas more advanced lesions become tethered and fixed with increasing depth of penetration.
  - Benefits of TRUS versus MRI:
    - TRUS is superior in assessing early T stage (T1/T2) cancers. TRUS offers excellent delineation of the relationship between a tumour, the mucosa, the muscularis propria, and tumour extension beyond the muscularis propria
    - MRI is superior to TRUS:
      - for assessment of the circumferential resection margin to determine whether it is threatened by tumour
      - for providing a larger field of view and more detail about proximal tumours
      - as it tends to be less operator and technique dependent
      - as it allows assessment of obstructing cancers (TRUS not possible)
      - in practice, the information obtained from TRUS and MRI is often complementary and, at many institutions, both procedures are done pre-operatively particularly for patients whose tumours extend beyond T2. For early staging of T1 and T2 lesions, TRUS offers excellent delineation of the relationship between a tumour, the mucosa, the muscularis propria, and tumour extension beyond the muscularis propria. However, TRUS is clearly limited in terms of tumours that extend deeply into the pelvic side-wall, into additional pelvic structures, or for the detection and characterisation of adenopathy in the internal and external iliac distribution.
  - Indications for neo-adjuvant treatment in rectal cancer:
    - some controversy exists and the indications are constantly changing
    - employed in:
      - resectable cancers to reduce local recurrence rates (typically by 50%) — usually short-course pre-operative radiotherapy (SCPRT)
      - non-resectable tumours to achieve ‘down-staging’ prior to surgery — usually long course chemoradiotherapy.
    - Large RCTs suggest 50% local reduction rates with a policy of routine short course pre-operative radiotherapy compared to selective post-operative radiotherapy (UK MRC07, Lancet 2009) for all tumour stages and heights. However, benefit is most pronounced for:
      - T3/4 tumours
      - clinically node-positive T1/2 tumours
      - more distal rectal tumours (mid- and lower-third tumours)
      - men
• anterior tumours
• when the circumferential resection margin is threatened; that is, cancer invading or are in close proximity to the mesorectal fascia on pre-operative imaging.
• Difference between short-course and long-course radiotherapy
  • SCPRT gives 25 Gy over 5 days and is followed by immediate surgery
  • long-course radiotherapy gives 50 Gy over 5 weeks and is combined with chemotherapy (infusion of 5-FU for 5 days on weeks 1 and week 5), then surgery 6–8 weeks post-treatment
  • the advantage of pre-operative radiotherapy is that the pelvic anatomy is undisturbed and there is less chance of the small bowel getting irradiated. The tissues are likely to be well oxygenated which increases radiosensitivity. It also enables assessment of the tumour response on histopathology enabling prognostication and need for adjuvant treatment.
• Surgery for rectal cancer
  • Depends on the location of the tumour, its relationship to the anal sphincters and the ability to get an oncologically clear distal margin.
  • In general, a 5 cm margin of distal clearance is preferred for poorly differentiated cancers. However, for other differentiated rectal cancers a 1.5–2 cm margin of clearance has been shown to be oncologically safe.
  • In rectosigmoid or upper rectal cancers 5 cm of distal clearance is easily achievable, allowing construction of a colorectal anastomosis to the mid-third of the rectum.
  • In mid- and lower-third rectal cancers, total mesorectal excision is necessary for complete oncological control. As a result, most surgeons would accept a distal clearance of 1.5 to 2 cm to allow GI restoration. This is supported by the available evidence. Due to the leak rate of distal anastomoses, most surgeons defunction with a loop ileostomy.
  • Tumours adjacent to (where not possible to get adequate distal clearance) or involving the sphincter require abdominoperineal excision of the rectum, although sphincter-saving surgery, involving inter-sphincteric dissection is performed in specialist centres in selected cases.
  • In order to preserve continence, it is necessary to have good sphincter function and an adequate reservoir. With complete removal of the rectum a reservoir can be reconstructed by creating a 5–8 cm colonic J pouch. Straight coloanal anastomosis result in fairly poor function for up to 2 years and have been reported to have a higher leak rate than colonic pouch anastomosis. More importantly, there have been a number of studies that demonstrate that early function with a colonic J pouch is superior to straight coloanal anastomosis, particularly in the elderly with compromised anal sphincters.
  • Accordingly, the options are:
    • high anterior resection — for upper rectal tumours, that is within 10–15 cm of the anal verge
    • low anterior resection (± colonic J pouch ± loop ileostomy) — for mid to upper rectal tumours i.e. within 6 — 15 cm of the anal verge
    • ultra-low anterior resection (ULAR) (± colonic J pouch) and loop ileostomy — for tumours within the mid to low rectum in which distal clearance of 1 cm can be achieved
    • abdominoperineal excision of the rectum and anus — for low rectal tumours in which a distal clearance of 1 cm cannot be achieved or in which there is involvement of the anal sphincters.
3.3 Common medium cases

- High versus low ligation of the inferior mesenteric artery (IMA):
  - high ligation involves ligation of the IMA flush with the aorta; low ligation involves ligation of the IMA at the sacral promontory preserving the left colic artery
  - there is no evidence that high ligation confers a better oncologically result or cancer survival. However, most surgeons would perform a high ligation in order to achieve adequate length of the proximal bowel to perform a tension free colorectal anastomosis following low anterior resection/ULAR.

- Anatomy of the pelvic nerves (see 2.4.2).

- Indications for a defunctioning ileostomy:
  - patients who have had neo-adjuvant radiotherapy and an ULAR
  - anastomotic leak rates as high as 10–20% have been reported after ULAR
  - while a defunctioning ileostomy does not absolutely prevent an anastomotic leak, it usually minimises the degree of sepsis and avoid repeat surgical intervention should it occur
  - other cases where there is concern about the anastomosis; for example:
    - evidence of an air leak after anastomosis testing with air and water
    - incomplete donuts in the circular stapling device
    - tension, or if the blood supply was questionable
    - difficult dissection and anticipation of problems post-operative (relative).

3.3.3. Colon cancer

![Right hemicolecetomy specimen](image)

**Figure 3.3.3A**
Right hemicolecetomy specimen
As previously stated, colorectal cancer cases are common. You may be introduced to a man sitting in a clinic room as follows: ‘This is Mr Smith. Would you please take a history from him and perform an appropriate examination’.

The history

- Introduce yourself and ask the patient what has been going on. The patient may indicate that he had a bowel cancer removed 4 months ago.
- Try to maintain a structure to your history, using the framework above. Begin by orientating yourself.
- The presenting symptoms and history of presenting complaint:
  - presentation: asymptomatic after a positive faecal occult blood test or during surveillance colonoscopy. Symptomatic: bleeding per rectum (PR) and its detail; altered bowel habit (constipation/diarrhoea/both); abdominal pain (SOCRATES) or distension
  - associated symptoms of the diseased (GI) system: nausea; vomiting; jaundice
  - other associated symptoms — systemic: weight loss; anorexia; malaise; shortness of breath
  - other associated symptoms — anaemia: palpitations, weakness, SOB, CCF
  - investigations: colonoscopy/staging radiology (CT/MRI)/biopsy.
- treatment:
  - Was the patient admitted electively or acutely?
  - What procedure was performed?
  - Which part of the colon was removed (right, left, all)?
  - Were there any complications after the operation?
  - Current status of the patient (diet, mobility, bowel function, expected date of discharge).
  - Will adjuvant treatment be required (radiotherapy or chemotherapy)?
- Risk factor assessment:
  - personal or family history of polyps/colorectal cancer/inflammatory bowel disease.
  - Past medical history.
• Surgical history.
• Drug history and allergies.
• Family history — particularly of endometrial/gastric/ovary/urothelium (HNPCC).
• Social history: occupation, home circumstances, smoking tobacco and alcohol consumption.

The examination
• Preparation — see above:
  • position the patient flat with one pillow under the head
  • expose the abdomen from nipples to pubic symphysis but state that ideally you
    would like to expose the patient to mid-thigh.
• General examination from the end of the bed:
  • overall condition
  • drains, tubes, infusions, TPN, IDC, oxygen etc.
• Assessment for peripheral signs, unless instructed otherwise:
  • vital signs
  • hands: stigmata of gastrointestinal disease — anaemia/clubbing/ malnutrition.
  • head and neck: cachexia (wasting of temporalis)/jaundice/pallor/ lymphadenopathy.
• Examination of the abdomen:
  • inspection: moving with respiration; distension; scars; drains; stomas; dressings;
    bruising, inspect wounds for signs of infection. look at drains, stomas and urine
    bag if present
  • palpation: ask if there is any pain — soft; tender; organomegaly; ascites. Assess
    groin and incision for evidence of a cough impulse; palpate the testicles
  • percussion: not usually relevant post-operatively unless distended and you want
    to determine if it is gaseous or fluid (ascites/ileus); percussion for shifting dullness;
    fluid thrill
  • auscultation: listen in four quadrants for the character of borborygmi, bruits over
    the renal arteries, aorta and liver.
• Conclusion:
  • say that you would do a digital rectal examination
  • cover the patient
  • reposition the patient
  • thank the patient
  • clean your hands again
  • only now turn to face the examiners.

The discussion
• The examiners may ask you to tell them what you found. An example of a succinct
  summary for this case would be as follows, ‘Mr Smith is a 54-year-old man who had
  a colonic cancer detected on a screening colonoscopy following positive FOBT. He
  has a family history of colonic cancer. His brother died of colon cancer aged 44 and
  his father had two colon cancers treated in his 50s and 60s before he died of a heart
  attack. The history and physical examination suggest he underwent an uncomplicated
  laparoscopic right hemicolectomy. He was told it had spread to the lymph glands
  and he has had chemotherapy. He has one son and one daughter and has seen a
  genetic counsellor.
• Average risk of developing colonic or rectal cancer in Australia:
  • bowel cancer affects 1 in 12 before the age of 85 years (Cancer in Australia 2010.
    Australian Institute of Health & Welfare).
• Causes of colonic and rectal cancer:
  • certain conditions predispose to the development of colorectal cancer, including IBD, acromegaly, history of uretero-colostomy and radiation therapy also increase risk
  • increasing age is a strong risk factor
  • it is a multi-factorial disease; in approximately 5%, there is an overwhelming genetic/inherited contribution (HNPCC/FAP — see below)
  • there are well-defined environmental factors, the most important are probably dietary
  • there is a correlation of consumption of total fat, saturated fat and cholesterol with increased cancer risk
  • dietary fibre is associated with reduced risk — it increases bulk to dilute potential carcinogens, speeds their transit through the colon, binds certain mutagens and favourably changes faecal pH
  • lack of physical activity, diabetes, increased concentration of insulin and IGF1 are associated with increased risk. There is a modest association with smoking.

• What is HNPCC?
  • It is an autosomal dominant inherited condition associated with an increased risk of colorectal cancer. Microsatellite instability (MSI) is the hallmark of HNPCC tumours. It was previously known as Lynch syndrome.
  • It is due to germ-line mutations in mismatch repair genes. The involved genes include hMLH1, hMSH2, and hMSH6. hMSH2 and hMLH1 and together account for > 90% of identifiable mutations.
  • 20% of cases are spontaneous germ-line mutations with no family history.
  • HNPCC is diagnosed on the basis of genetic testing. Given the expense involved, individuals fulfilling the Amsterdam criteria and tumours meeting the Bethesda criteria are tested.
    • Amsterdam criteria can be remembered by ‘3, 2, 1 rule’; that is, three relatives affected with an HNPCC cancer (colorectal, endometrium, small bowel, urothelium), over two successive generations, with one cancer under the age of 50 years. These criteria are neither sensitive (79%) nor specific (61%) for the diagnosis.
    • The Bethesda criteria are used to determine whether to test for MSI. They can be remembered using Bethesda:
      • bet
      • histopathological characteristics of MSI in pts <60 yrs
      • extra HNPCC cancers (synchronous/metachronous)
      • single (≥1) 1st degree relative <50 yrs
      • double (≥2) 1st/2nd degree relative HNPCC cancer
      • age <50 yrs.
    • Tumours can be MSI-high (> 2 loci show band-shifts), MSI-low (1 locus shows band shifts), MS-stable (no band shifts). Almost all HNPCC cancers and about 70% of adenomas are MSI-high. However 15% of sporadic colorectal cancers are MSI-High.
    • Immunostaining for loss of expression of hMLH1, hMSH2, hMSH6 or hPMS2 may be an indicator of a germ-line mutation in the gene coding for that protein and has 76% sensitivity in predicting MSI status and is 100% specific. Diagnosis by germ-line genetic testing allows identification of the causative germ-line mutation in an affected individual. This also means that other at-risk family members can be offered predictive testing.
3.3 Common medium cases

- Investigation and staging of a patient with colon cancer
  - Blood tests:
    - FBC, EUC, LFTs, CEA.
  - Colonoscopy:
    - for (histopathological) diagnosis
    - to exclude synchronous colonic neoplasms
    - barium enema/virtual colonography remain alternatives.
  - CT scan chest, abdomen and pelvis:
    - assessment of tumour (local advanced) and complications
    - staging to detect metastatic disease.

- Management of colon cancer
  - Stage the cancer.
  - Determine intent (curative/palliative).
  - Determine patient’s wishes.
  - Assess patient’s fitness for surgery.
  - Discuss in MDT setting (if elective presentation).
  - Discuss operative approach (Laparoscopic vs Open colectomy).
  - Complete clinicopathological staging post-operatively to determine need for adjuvant therapy:
    - stage III cancers and high risk stage II disease (T4, poor histologic grade, peri-tumoural lymphovascular involvement, obstruction, T3 with local perforation, and close or positive margin).

- Staging of colon cancer — TNM AJCC (clinicopathological staging)
  - Primary tumour (T):
    - Tis, carcinoma in situ: intraepithelial or invasion of the lamina propria
    - T0, no evidence of primary tumour
    - T1, tumour invades submucosa
    - T2, tumour invades muscularis propria
    - T3, tumour invades through the muscularis propria into the subserosa or into non-peritonealised pericolic or perirectal tissues
    - T4, tumour directly invades other organs or structures and/or perforates visceral peritoneum
  - Regional lymph nodes (N):
    - N0, no regional lymph node metastasis
    - N1, metastasis in one to three regional lymph nodes
    - N2, metastases in four or more regional lymph nodes
  - Distant metastasis (M):
    - M0, no distant metastasis
    - M1: distant metastasis.

- 5-year survival rate:
  - stage I (T1/2, N0) 85–90%
  - stage II (T3/4,N0 66%
  - stage III (N+) 40–50%
  - stage IV (M+) 0–10% (increased to 30% if metastectomy).

- Evidence for laparoscopic compared to open colectomy
  - Surgery is the only potentially curative modality of treatment — the aims must be the same, irrespective of the approach:
    - achieve an R0 resection by resecting the affected segment of bowel with at least 5 cm of proximal and distal clearance along with the lymphovascular drainage
    - restore bowel continuity if possible.
Laparoscopic surgery has been shown to be non-inferior to open resection in three large RCTS (COST/CLASSIC/COLOR). Laparoscopic surgery may be superior in terms of: (a) decrease length of stay; (b) quicker return of bowel function; and (c) less post-operative pain.

What is the strategy of treatment for primary colon cancer?

Surgery for primary colonic cancer requires removal of primary tumour with adequate margins and regional lymphadenectomy with restoration of the colonic continuity.

The nature of the segmental colonic resection required depends on the location of the primary. Surgery involves resection of the affected colonic segment with its lymphovascular pedicle, resulting in an en bloc lymphadenectomy. This ligation of the artery will in turn usually determine the minimum amount of bowel that must be resected to leave well vascularised bowel for anastomosis.

The resections for a right-sided cancer include right hemicolectomy or extended right hemicolectomy (hepatic flexure cancers). Transverse colon cancers can be treated by extended right hemicolectomy, left hemicolectomy or subtotal colectomy (Note: transverse colectomy is not recommended as it is not oncologically and blood supply and tension issues can increase rate of anastomotic leak. Left sided lesions can be treated by extended left hemicolectomy, high anterior resection or Hartmann’s procedure. Synchronous lesions can be treated with synchronous resections (and 2 anastomoses), although subtotal colectomy is preferred, particularly in the setting of HNPCC/mucosa field change.

Role of adjuvant treatment of colon cancer

The goal is to eradicate micrometastases, reducing the likelihood of disease recurrence and distant metastases.

30–70% of stage III (node-positive cancers) patients develop recurrence and its use is best established in such patients.

Standard (5FU/folinic acid) chemotherapy confers a survival benefit of 5–6% in node-positive cancers (Dukes C, stage III) compared to no adjuvant therapy.

However, a (unidentifiable) proportion will be cured by surgery alone and will only be exposed to the risks of chemotherapy without benefit. Additionally, a (unidentifiable) proportion will relapse despite chemotherapy and will succumb to their disease.

The MOSAIC trial (NEJM 2004) suggested improved survival advantage with addition of Oxaliplatin to standard regime of 5FU/folinic acid, giving rise to FOLFOX schedules (FOL—Folinic acid; F — Fluorouracil [5-FU]; OX — Oxaliplatin).

Current recommendations state that all medically appropriate node-positive patients receive adjuvant chemotherapy for 6 months after resection.

Possible benefit to high-risk stage II (debated): (a) T4 tumours; (b) poor histologic grade; (c) peritumoural lymphovascular involvement; (d) obstructing tumour; (e) T3 with local perforation; (f) close or positive margins.

However, standard chemotherapy confers an absolute survival advantage of 1.5% (UK QUASAR II) and FOLFOX confer an absolute survival advantage of 3% in node-negative (Dukes B) cancers.

Radiation therapy has no defined role in current guidelines for treatment of colon cancer.

Median survival is increased from a median of 6 months (no chemotherapy) to 12 months with standard regimens and up to 18–20 months with FOLFOX in non-resectable stage IV patients (metastatic disease). However, the response rate is only 20%.
• Complications of colon cancer and their surgical treatment
  • Perforation, obstruction and bleeding are the most common complications of colon cancers requiring emergency treatment
  • Obstructing lesions in the distal colon (and rectum) can have deleterious effects on the proximal/entire colon (the caecum is particularly vulnerable) if the ileo-caecal valve is competent and a closed loop obstruction ensures. This can result in caecal ischaemia, compromise, damage and perforation. This will change the operation which is necessary.
• Hepatic metastases (for assessment see 2.4.20; for selection and treatment see 3.3.7).
• Colonic polyps, Haggit’s levels, adenoma-carcinoma sequence, FAP (see 4.6.2).

<table>
<thead>
<tr>
<th>OPTION</th>
<th>INDICATION</th>
<th>ADVANTAGE</th>
<th>DISADVANTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Defunctioning proximal colostomy without resection</td>
<td>High risk patient Elderly or frail Palliation</td>
<td>No anastomosis</td>
<td>Burden of colostomy. Primary resection should be performed whenever possible</td>
</tr>
<tr>
<td>Hartmann’s procedure</td>
<td>High risk patient. Inexperienced surgeon</td>
<td>No risk of anastomosis</td>
<td>Need for further surgery Stoma never reversed in &gt; 50%</td>
</tr>
<tr>
<td>Resection with on-table colonic irrigation and primary anastomosis</td>
<td>Allows one stage resection in unprepared bowel</td>
<td>Addresses the issue of bowel preparation</td>
<td>Cannot use if damage to proximal colon Increased operating time</td>
</tr>
<tr>
<td>Colonic stent</td>
<td>Palliation, bridge to surgery</td>
<td>Lower colostomy rate, allows elective resection</td>
<td>Uncertain long term outcome in bridge to surgery group Expense Limited availability Technical failure rate</td>
</tr>
<tr>
<td>Subtotal colectomy</td>
<td>Synchronous tumours and proximal bowel damage</td>
<td>As safe as segmental resection</td>
<td>Suboptimal functional outcome Extensive surgery</td>
</tr>
</tbody>
</table>

3.3.4 Ulcerative colitis

![Figure 3.3.4](https://example.com/figure3.3.4)

Ulcerative colitis

Sample proofs @ Elsevier Australia
Such cases are likely to be post-operative, but an inpatient or outpatient with UC who is booked for, or contemplating, elective surgery may be presented.

Extent of disease is usually:
- proctitis 50%
- proctocolitis 30% (extends to left colon)
- extensive colitis 20% (beyond splenic flexure).

The history
- Introduce yourself and ask the patient what has been going on.
- Try to maintain a structure to your history, using the framework above. Begin by orientating yourself.
- The presenting symptoms and history of presenting complaint:
  - presentation: cardinal symptoms of abdominal pain and bloody diarrhoea. Abdominal pain (SOCRATES) and distension (toxic dilatation). Bleeding per rectum (PR) and diarrhoea — amount of blood, stool frequency, relative amounts of blood/stool. Mucous discharge per rectum; faecal incontinence; tenesmus
  - associated symptoms of the diseased (GI) system: nausea; vomiting; jaundice (PSC)
  - other associated symptoms — systemic: weight loss; anorexia; malaise; anaemia
  - other associated extracolonic manifestations:
    - urinary: calculi
    - skin: erythema nodosum/multiforme, pyoderma gangrenosum
    - musculoskeletal: arthritis, ankylosing spondylitis — independent of IBD activity
    - eyes: uveitis, chorioretinitis, iridocyclitis
  - investigations: bloods/radiology/colonoscopy/biopsy.
  - treatment:
    - if the patient is post-operative, what was the indication for surgery?
    - any acute severe attacks (toxic dilatation, perforation, bleeding, unresponsive to medical treatment)?
    - chronic active disease problems: steroid dependence/failed medical treatment/recurrent acute attacks, growth retardation, dysplasia associated lesion or mass (DALM)/malignancy?
    - if the patient is post-operative, what operation was performed:
      - acute attack — total colectomy/mucous fistula and end ileostomy (about 65%)
      - chronic disease — (a) total colectomy/mucous fistula and end ileostomy; (b) proctocolectomy and end ileostomy; or (c) proctocolectomy and ileal pouch anal anastomosis
      - were there any complications after the operation
      - current status of the patient (diet, mobility, bowel function, expected date of discharge)?
  - Past medical history.
  - Surgical history.
  - Drug history and allergies.
  - Family history.
  - Social history: occupation, home circumstances, smoking tobacco and alcohol consumption.

The examination
- Preparation (see above):
  - position the patient flat with one pillow under the head
• expose the abdomen from nipples to pubic symphysis but state that ideally you
  would like to expose the patient to mid-thigh.
• General examination from the end of the bed:
  • overall condition — pale/jaundice/malnourished/Cushing appearance
  • drains, tubes, infusions, TPN, IDC, oxygen etc.
• Assessment for peripheral signs, unless instructed otherwise:
  • vital signs
  • hands: stigmata of gastrointestinal disease — anaemia/clubbing/malnutrition
  • head and neck: cachexia (wasting of temporalis)/jaundice/pallor/lymphadenopathy
  • eyes: uveitis, chorioretinitis, iridocyclitis
  • legs: erythema nodosum, pyoderma gangrenosum, arthritis of knees, ankylosing
    spondylitis.
• Examination of the abdomen:
  • inspection: moving with respiration; distension; scars; drains; stomas (mucus
    fistula); dressings; bruising. Inspect wounds for signs of infection. Look at stoma
    and urine bag if present
  • palpation: soft; tender; organomegaly; ascites. Assess groin and incision for evi-
    dence of a cough impulse. Palpate the testicles
  • percussion: not usually relevant post-operatively unless distended and you want
    to determine if it is gaseous or fluid (ascites/ileus). Percussion for shifting dullness;
    fluid thrill
  • auscultation: listen in four quadrants for the character of borborygmi, bruits over
    the renal arteries, aorta and liver.
• Conclusion:
  • say that you would do a digital rectal examination (depending if pre-op or post-op
    and no low anastomoses)
  • cover the patient
  • reposition the patient
  • thank the patient
  • clean your hands again
  • only now turn to face the examiners.

The discussion
• Confirmation of a diagnosis of ulcerative colitis
  • The diagnosis is made on the basis of clinical, luminal imaging (endoscopy/radiol-
    ogy) and pathology.
  • Investigations contribute to the diagnosis and also allow assessment of disease
    severity.
  • Full history and examination to exclude other causes of colitis (see 2.2.7 Colitis)
    and to establish GI and extra GI involvement to provide information of UC Vs
    Crohn’s (see below).
  • Stool culture sample to exclude infectious cause.
  • Bloods — FBC (anaemia/ leukocytosis), ESR, CRP, LFTs, (inc. Albumin).
  • Radiology — AXR for toxic dilatation and signs of inflammation (granularity, loss
    of haustra causing lead-pipe appearance).
  • Flexible sigmoidoscopy/colonoscopy — rectum is almost always involved. Features
    include loss of vascularity, mucosal oedema, granularity, erythema, contact bleed-
    ing and frank ulceration, pseudopolyps (from previous attacks and regeneration).
    Findings are usually confluent unlike Crohn’s disease which is characterised by
    skip lesions and patchy disease. Defines proximal extent of the disease.
- Biopsies — demonstrate mucosal and submucosal involvement, Crypt of Lieberkuhn abscesses, absence of (non-caseating) granulomas.
- Clinical and histopathological differences between UC and Crohn’s disease. (Please also refer to 4.6.3.)

| Table 3.3.4 Clinical and histological features of ulcerative colitis and Crohn’s disease |
|-----------------------------------------------|-----------------------------------------------|
| **Clinical Feature**                          | **ULCERATIVE COLITIS**                         | **CROHN’S DISEASE** |
| Distribution                                  | Colon and rectum                              | Entire GI tract     |
| Appearance                                    | Superficial and disease areas are confluent   | Cobblestone appearance — linear deep fissuring ulcers (serpiginous) with islands of oedematous mucosa in between Inflammatory polyps |
| Rectal involvement                            | Always                                        | Rarely              |
| Strictures                                    | Rarely                                        | Common              |
| Fistulae                                      | Never                                         | Common              |
| Anal involvement                              | Rare                                          | Common              |
| Malignancy risk                               | 10% at 20 yrs                                 | 10% at 20 yrs       |
| **Histological**                              | **Bowel wall**                                | **Non-caseating granulomas** |
|                                              | Mucosa and submucosa                          | Absent              |
|                                              | Full thickness                                | Present 60–70%      |
|                                              | Crypt abscesses                               | Commons             |
|                                              | Rare                                          | Common              |
|                                              | Goblet cell                                  | Depleted            |
|                                              | Preserved                                     |                     |

- Extra-intestinal manifestations and intestinal complications of UC/IBD
  These can be easily remembered using the acronym **ULCERATIVE COLITIS**.
- Extra-intestinal manifestations:
  - **U** Urinary calculi: especially oxalate (Crohn’s disease)
  - **L** Liver: cirrhosis, sclerosing cholangitis, fatty liver
  - **C** Cholelithiasis: decreased bile acid resorption
  - **E** Epithelium: erythema nodosum/multiforme, pyoderma gangrenosum
  - **R** Retardation of growth and sexual maturation — especially in kids
  - **A** Arthralgias — arthritis, ankylosing spondylitis — independent of IBD activity
  - **T** Thrombophlebitis — migratory
  - **I** Iatrogenic: steroids, blood transfusions, surgery
  - **V** Vitamin deficiencies
  - **E** Eyes: uveitis, chorioretinitis, iridocyclitis
- Intestinal complications:
  - **C** Cancer: increased by long duration/early onset, pancolitis
  - **O** Obstruction: rare with UC, common in Crohn’s especially post-surgery
  - **L** Leakage (perforation): can form abscess especially in Crohn’s (20%)
  - **I** Iron deficiency: haemorrhage
  - **T** Toxic megacolon: 3% (more often in UC)
  - **I** Inanition: severe wasting due to malabsorption and decreased oral intake
  - **S** Stricture/fistulas (40% of Crohn’s), perianal abscesses

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3.3 Common medium cases

- Principles of treatment of ulcerative colitis
  - Depends on the severity of disease.
  - The majority of cases are treated medically unless complications arise. 70% of patients with UC are managed medically. 30% of patients will require surgical intervention that is usually curative.
  - The goal of treatment is to induce remission and maintain disease in this state. IBD needs to be managed in collaboration with a gastroenterologist/nutritionist/stoma therapist when and where indicated.
  - Assessment of severity — severe disease Truelove and Witt criteria:
    - no bloody stools/day > 6
    - pulse rate > 90
    - temperature > 37°
    - haemoglobin < 105 g/L
    - ESR/CRP > 30
  - Treatment of acute severe ulcerative colitis
    - Medical treatment:
      - admit to hospital
      - resuscitate and correct electrolytes; nutritional support
      - IV hydrocortisone 100 mg QDS
      - stop NSAIDs, anticholinergics, antidiarrheals (increase toxic dilatation)
      - transfuse PRC to keep Hb > 100 g/L
      - aim maximum 5/7 therapy — 70% response rate
      - serial clinical/serological/radiological examinations to detect complications
      - unresponsive:
        - 2nd line: cyclosporine/tacrolimus or infliximab
        - colectomy.
    - Surgical treatment:
      - indications — BUMP:
        - bleeding
        - unresponsive to medical Rx
        - megadilation (toxic dilation)
        - perforation
      - operation of choice: subtotal colectomy, ileostomy ± mucus fistula.
  - Treatment of chronic ulcerative colitis
    - Medical treatment:
      - proctitis: topical 5-ASA (mesalzine 1G OD). Escalate to addition of topical steroid or oral 5-ASA
      - left-sided disease: topical 5-ASA and oral mesalazine (2G/day)
      - no response — oral steroids with a reducing dose over 8/52
      - extensive disease: as for left-sided but lower threshold for use of steroids.
    - Surgical treatment:
      - indications — 3Ms:
        - malignancy/severe dysplasia/DALM
        - medical treatment failure
        - maturation (growth/nutrition) failure
      - operations to consider:
        - the 'gold standard' is a panproctocolectomy and permanent ileostomy formation
        - restorative proctocolectomy and ileal J-pouch anal anastomosis (IPAA) for those who wish to avoid a stoma.
• colectomy with ileorectal anastomosis in highly selected cases with relative rectal sparing
• colectomy with ileostomy and rectal preservation (so IRA is possible later).

- Complications of IPAA — 4Ps
  • Pouch failure occurs in 5–10%. Reasons include:
    • pelvic sepsis (50%) from leak or infected haematoma
    • poor function (30%)
    • pouchitis (10%).
  • Pouchitis — aetiology is unknown but related to the original disease process, symptoms include frequency and urgency of stool. Need endoscopic confirmation of inflammation to confirm. Treatment is antibiotics (metronidazole, ciprofloxacin or augmentin). Long-term effects of chronic pouchitis predisposes to dysplasia.
  • Post-operative complications — 4Ss:
    • small bowel obstruction
    • sepsis (pelvis)
    • stricture of anastomosis — occurs in 5–20%; may require surgical intervention if intestinal obstruction (dilatation/revision)
    • sinus — pouch vaginal/perineal.
  • Poor function
    • normal function is 6–8 bowel motions per day; however, nocturnal frequency and incontinence are better markers of poor function. Trial of antidiarrheals should be given to improve function. There is a tendency for function to improve over time.

3.3.5 Oesophageal carcinoma

The history
• Introduce yourself and ask the patient what has been going on. The patient may indicate that he has had or is some way through treatment for oesophageal cancer.
• Try to maintain a structure to your history, using the framework above. Begin by orientating yourself.
• The presenting symptoms and history of presenting complaint:
  • presentation: dysphagia (difficulty) or odynophagia (pain) with full quantification (liquids/semi-solids/solids) and progression; haematemesis; previous reflux; abdominal pain (SOCRATES) or distension
  • associated symptoms of the diseased (GI) system: nausea; vomiting; jaundice
  • other associated symptoms — systemic: weight loss; anorexia; malaise
  • other associated symptoms — chest: SOB; cough; pneumonia
  • investigations: upper GI endoscopy/barium swallow; staging radiology (CT/MRI/PET scan)/endoluminal ultrasound/biopsy.
• treatment:
  • Was a staging laparoscopy performed?
  • Was neo-adjuvant treatment required or discussed?
  • What surgery was performed? Separate neck/chest/abdomen incisions?
  • Were there any complications after the operation?
  • Current status of the patient (diet, mobility, bowel function, expected date of discharge).
  • Will adjuvant treatment be required (radiotherapy or chemotherapy)?
3.3 Common medium cases

- Risk factor assessment:
  - personal history of preceding Barrett’s disease.
- Past medical history.
- Surgical history.
- Drug history and allergies.
- Family history.
- Social history: occupation, home circumstances, smoking tobacco and alcohol consumption.

The examination

- Preparation (see above):
  - permission — ask to examine the patient’s neck, chest and abdomen
  - position the patient flat with one pillow under the head
  - expose the neck, chest and abdomen (to pubic symphysis but state that ideally you would like to expose the patient to mid-thigh).
- General examination from the end of the bed:
  - overall condition
  - drains, tubes, infusions, TPN, IDC, oxygen etc.
- Assessment for peripheral signs, unless instructed otherwise:
  - vital signs
  - hands: stigmata of gastrointestinal disease — anaemia/clubbing/ malnutrition
  - head and neck: neck scars and supraclavicular lymphadenopathy; cachexia (wasting of temporalis)/jaundice/pallor.
- Examination of the chest/abdomen:
  - inspection: look for previous scars and presence of a portacath — get the patient to point out scars. (Note: most patients will have a right thoractomy and midline laparotomy. Some will also have a left-sided neck incision; patients who have had a minimally invasive oesophagectomy may have port site scars that are hard to see); drains; dressings; bruising. Inspect wounds for signs of infection
  - palpation: soft; tender; organomegaly — especially the liver edge; ascites. Assess groin and incision for evidence of a cough impulse. Palpate the testicles
  - percussion: not usually relevant post-operatively unless distended
  - auscultation: listen to the chest and all four quadrants in the abdomen for the character of borborygmi, bruits over the renal arteries, aorta and liver.
- Conclusion:
  - say that you would do a digital rectal examination in the pre-operative setting (distance of cancer from the anal verge; prostate; sphincter tone)
  - cover the patient
  - reposition the patient
  - thank the patient
  - clean your hands again
  - only now turn to face the examiners.

The discussion

- The examiners may ask you to tell them what you found. An example of a succinct summary for this case would be as follows, ‘Mr X is a 54-year-old man who had a distal oesophageal adenocarcinoma detected on a screening endoscopy performed because of a history of Barrett’s oesophagus. He received pre-operative chemotherapy and underwent an uncomplicated Ivor-Lewis oesophagectomy. He was told it had spread to the lymph glands and he has had further post-operative chemotherapy.’
• Investigation of a patient with symptoms suggestive of oesophageal malignancy:
  • bloods: FBC (anaemia), iron studies (iron deficiency), CEA (baseline for follow-up)
  • upper GI endoscopy:
    • the endoscopy should focus on definition of the location of the lesion as precisely as possible, using both distance from incisors and internal and external landmarks
    • biopsy is essential to differentiate adenocarcinoma and squamous cell carcinoma as the management can be very different
    • a complete survey for other lesions and biopsy of mucosa around the lesion may give information about adjacent non-invasive disease
  • endoscopic US provides valuable information about the depth of invasion of the primary and potential regional nodal metastasis
  • CT of the neck, chest, abdomen and pelvis is essential
  • FDG-PET — will up-stage patients (who seem to be candidates for surgery on all other grounds) by about 25% by detection of occult systematic metastasis. This may help avoid resection in these patients
  • the greatest accuracy of staging appears to be achieved when CT is combined with EUS (Table 3.3.5)
  • for those with tumours of the middle or lower third of the oesophagus, diagnostic laparoscopy will detect occult peritoneal or liver disease in a small percentage.

<table>
<thead>
<tr>
<th>MODALITY</th>
<th>T STAGE</th>
<th>N STAGE</th>
<th>M STAGE</th>
<th>OVERALL STAGING ACCURACY</th>
</tr>
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<tr>
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<td>40</td>
<td>50</td>
<td>85</td>
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<td>EUS</td>
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<tr>
<td>CT + EUS</td>
<td>90</td>
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</tr>
</tbody>
</table>

• What is the epidemiology and aetiology of oesophageal malignancy?
  • The disease is five times more common in men with a peak age of presentation of 50–70. There are marked geographic variations in the incidence of oesophageal cancer.
  • Oesophageal cancer is really two different disease entities with different epidemiology and management.
  • SCC is predominant worldwide (90%), but adenocarcinoma is more common (60%) is the Western world.
  • SCC is linked with excess alcohol intake, smoking, HPV (16 and 18). Predisposing factors include: achalasia (6–30x risk), alkaline stricture (22x risk), peptic stricture, Plummer Vinson syndrome (web in post-cricoid region and iron-deficient anaemia), Zenker’s diverticulum.
  • SCC is located predominantly in the middle third (50%), with about 35% in the lower third and 15% in the upper third of the oesophagus.
  • The vast majority of adenocarcinoma are found in the lower third of the oesophagus. Adenocarcinoma has a rising incidence in industrialised nations. It has epidemiological similarity cancer of the cardia of the stomach.
  • The predominant aetiological factor is gastro-oesophageal reflux, which leads to metaplastic change with progressive dysplasia and eventual adenocarcinoma.
• The risk of oesophageal adenocarcinoma is 30–50x higher with Barrett’s oesophagus.
• Tumours around the OGJ have been classified into three types (Siewart):
  • type I tumours are adenocarcinoma of the distal oesophagus usually arising from Barrett’s oesophagus which may have infiltrated the GOJ junction from above
  • type II lesions are true carcinoma of the cardia arising from the cardiac epithelium or short segments with intestinal metaplasia at the oesophago-gastric junction
  • type III junctional cancers are subcardial gastric carcinoma, which infiltrates the oesophagogastric junction and distal oesophagus from below
  • the type II cancers have demographic and pathological features similar to the adenocarcinoma found in Barrett’s oesophagus. Compared to type III cancer they display aggressive behaviour, worse prognosis, greater propensity to serosal invasion and lymph node metastasis at presentation and not being associated with atrophic gastritis or intestinal metaplasia of the stomach.
• How is oesophageal cancer staged?
  • The AJCC (7th edition) staging system for oesophageal and junctional tumours has been recently updated (Ann Surg Oncol (2010) 17:1721–1724). This classification pertains to oesophageal tumours and tumours of the GOJ. It includes both SCC and Adenocarcinoma. The junctional tumour group includes ’cancers whose epicentre is in the distal thoracic oesophagus, esophagogastric junction, or within the proximal 5 cm of the stomach (cardia) that extend into the esophagogastric junction or distal thoracic oesophagus (Siewert III)’.
  • The upper, middle and lower thirds of the oesophagus are defined according to distance from the incisor teeth at 20–25 cm, 25–30 cm and 30–40 cm respectively.
  • T stage:
    • Tx primary tumour cannot be assessed
    • T0 no evidence of primary tumour
    • Tis high grade dysplasia
    • T1 tumour invades lamina propria or submucosa
    • T2 tumour invades muscularis propria
    • T3 tumour invades adventitia
    • T4a: resectable cancer invades adjacent structures such as pleura, pericardium, diaphragm
    • T4b: non-resectable cancer invades adjacent structures such as aorta, vertebral body, trachea.
  • N stage:
    • Nx regional lymph nodes cannot be assessed
    • NO no regional lymph node metastasis
    • N1 1 to 2 positive regional lymph nodes
    • N2 3 to 6 positive regional lymph nodes
    • N3 7 or more positive regional lymph nodes.
  • The definition of a regional lymph node metastasis now includes any periesophageal node from cervical to celiac, irrespective of the location of the primary.
  • M stage:
    • Mx distant metastasis cannot be assessed
    • M0 no distant metastasis
    • M1 distant metastasis.
Principles of oesophageal cancer treatment

- Treatment is dictated by disease-related factors (mainly stage) and patient-related factors (fitness for surgery and wishes).
- Due to improved surveillance of Barrett’s oesophagus, early cancers are increasingly being recognised. Endoscopic mucosal resection (EMR) is generally an acceptable definitive treatment with cancer not entering the submucosa (Tis and T1a), as there is a rate of lymph node metastasis (<3%) lower than the mortality of oesophagectomy. However, where the EMR specimen shows invasion into the submucosa (T1b), the risk of lymph node metastasis is higher (15–20%) and surgery is generally recommended for a fit patient.
- A patient should only be considered for surgery if there is a reasonable chance of resection without excess morbidity or mortality (not invading aorta, vertebra or heart) and there is likely to be oncological benefit. For patients with systemic metastasis (such as liver, lung or bone), peritoneal disease or lymphatic metastasis outside of the field of resection (such a supraclavicular metastasis in a GOJ cancer), the disease course is unlikely to be altered by resection of the primary and peri-oesophageal lymph nodes. For these patients, in addition to those who are unfit for or decline surgery, palliative treatments are indicated.

Multimodal treatment of oesophageal cancer

- For patients who are to undergo surgery, the standard practice in Australia is to offer pre-operative chemotherapy (5-FU and Cisplatin) according to the MRC OEO2 (MAGIC) protocol. If feasible, this is then followed further post-operative chemotherapy.
- Generally there are few trials (except MRC OEO2) examining post-operative adjuvant chemotherapy. The absolute survival benefit of this protocol is 5–10% compared with surgery alone. The benefit is similar for SCC and adenocarcinoma.
- There is little evidence to support radiotherapy (RT) alone in the adjuvant or neo-adjuvant setting. However, neo-adjuvant chemo-RT may be offered in stage II and III oesophageal cancer. The potential advantages include, down-staging and debulking, earlier treatment of micrometastases, decreased tumour seeding at surgery and increased radio-sensitivity due to increased tumour oxygenation before surgery. Also there is the potential for biological selection (avoiding surgery for patients who progress on treatment) so avoiding unnecessary radical surgery.
- Neo-adjuvant Chemo-RT trials show an absolute survival advantage in the order of 10–15% when compared to surgery alone. SCC of the proximal third of the oesophagus is generally treated with definitive chemo-RT (like a head and neck primary). For SCC anywhere in the oesophagus, chemo-RT appears to produce similar results to radical surgery.
- For patients who have locally advanced tumours, so-called trimodality treatment is an option. Here chemo-RT is followed by surgery if the tumour is down-staged. This appears to offer a survival advantage over chemo-RT alone.
- Clearly all these treatment options need to be discussed in a multidisciplinary team meeting and individualised according to the appropriate patient and disease-related factors.

Surgical treatment of oesophageal cancer

- Subtotal oesophagectomy in all patients with SCC/adenocarcinoma of middle and lower third as extensive submucosal spread.
- The general principles of oesophagectomy for cancer require abdominal and mediastinal lymphadenectomy. This provides superior staging and there is some evidence that it improves loco-regional control.
3.3 Common medium cases

- Splenectomy may be performed in patients with junctional adenocarcinoma, although there is no evidence that this is of benefit.
- Reconstruction using the stomach as a conduit generally gives best functional outcomes and there is increased mortality associated with colonic or small bowel conduits.
- Generally, vagus-sparing oesophagectomy, without lymph node dissection, is used only with early tumours confined to mucosa (not reaching the muscularis mucosae) diagnosed by EMR. This is not standard and many of these tumours would now be treated by EMR.
- There is no evidence for benefit of routine three-field nodal dissection.
- Feeding jejunostomy is best option for post-operative nutrition.
- There are four commonly used options in open oesophageal resection for cancer.
  1. The left thoracoabdominal (Sweet) approach involves an incision in the sixth intercostals space with division of diaphragm. It offers excellent exposure of GOJ but access to the proximal oesophagus is limited by the arch of aorta.
  2. The laparotomy and right thoracotomy (Ivor-Lewis) procedure is the most commonly used in Australia. Through the abdominal incision the stomach is mobilised, the cardia is resected (along with the left gastric artery). A gastric conduit is fashioned where the stomach survives on the right gastric and/or gastro-epiploic artery. The exposure of the GOJ from the chest is limited, but the exposure of rest of thoracic oesophagus is excellent. Generally the azygous vein is divided to allow access to oesophagus.
  3. The transhiatal (Grey-Turner) oesophagectomy (as popularised by Orringer) involves an incision in the abdomen and neck only. The oesophagus is mobilised by blunt dissection in posterior mediastinum from neck and abdomen. The nodes in the upper and middle third of mediastinum cannot be removed en bloc. There is an increased risk of damage to bronchus and azygous vein with tumour of the upper and middle third. Whilst this procedure does avoid a thoracotomy, and can be used in a patient with poor lung function, it probably achieves inferior oncological staging (if not results) due to the inability to dissect the mediastinal nodes en bloc.
  4. Finally the McKeown procedure is conducted as an Ivor-Lewis but includes an incision in the neck through which the anastomosis is fashioned. Although initially proposed as a means of circumventing an intra-thoracic leak (should one occur), the anastomosis usually falls back into the thoracic inlet and still causes mediastinitis if leak occurs.

- Palliative treatment of oesophageal cancer
  - Most patients either present with disease too advanced for surgery or are unfit for surgery. Therefore the main challenge in care of oesophagus cancer is effective palliative treatment.
  - A palliative pathway is indicated if the patient so desires after being fully informed or if they are not fit for surgery or definitive chemo/RT. Equally palliation is required if they have clear evidence of systemic spread or non-respectability.
  - Both localised disease and metastatic symptoms may require attention. Localised symptoms include dysphagia, odynophagia, haematemesis, cough, chest pains, reflux hoarseness and chronic bleeding leading to anaemia. Metastatic symptoms include anorexia, weight loss, fatigue, upper abdominal pain and constipation. The options for treatments are set out below.
  - Palliative chemotherapy: there is little evidence to suggest that this is superior to best supportive care.
• Palliative external beam radiotherapy: achieves palliation of dysphagia in < 40% of patients and has largely been superseded by brachy therapy or multi-modality treatment.
• Palliative brachy therapy: this treatment consists of a single dose intracavity radiation (12Gy). Brachy therapy has been shown to offer similar palliation to metal stents with fewer complications (bleeding, perforation) and should be considered as a primary mode of palliation for patients with a life expectancy greater than 3 months.
• Self expanding metallic stents (SEMS): SEMS have superseded intubation with rigid prostheses because of greater efficacy and fewer complications.
• Neodymium — yttrium aluminium garnet (YAG) laser: YAG laser is useful for temporary relief of dysphagia before surgery or definitive palliation with SEMS or Brachy therapy. It useful for tumour ingrowth or overgrowth over a SEMS. It is useful for the cervical oesophagus where stent placement is impossible, but may require multiple treatments. Argon coagulation has same advantages as laser but is cheaper and more available.

3.3.6 Chronic liver disease

In the fellowship examination, patients with chronic liver disease have usually had an operation for another indication and the background history of chronic liver disease is an issue in the peri-operative setting. In this example we will describe a patient who has had surgery for a strangulated umbilical hernia.

The evaluation of a patient with suspected chronic liver disease should include:
• diagnosis of the cause of liver disease
• determination of the functional hepatic reserve
• definition of the portal venous anatomy and hepatic haemodynamic evaluation, and
• localisation of the site of upper gastrointestinal haemorrhage, if present.

The history
• Introduce yourself and ask the patient what has been going on. The patient will indicate that he needed an emergency operation for a lump at the umbilicus.
• Try to maintain a structure to your history, using the framework above. Begin by orientating yourself.
• The presenting symptoms and history of presenting complaint:
  • presentation of lump: when first appeared, how noticed, changes since then (bigger?), any other lumps, does/did it disappear when laying flat
  • symptoms of complications: incarceration — irreducible; strangulation — pain, skin changes, unwell; obstruction — nausea, vomiting, abdominal pain/distension
  • associated symptoms of liver disease: weight loss; anorexia; malaise; jaundice (painful/painless), pale stools, dark urine, bruising, bleeding (haematemesis, melaena, per rectum), abdominal distension, leg oedema
  • previous investigations/interventions/treatments.
• Risk factor assessment:
  • chronic alcoholism, hepatitis, complicated biliary disease
  • tattoos, IVDU, blood transfusion, contact with rats (Farm)
  • exposure to hepatotoxins.
• Past medical history.
• Surgical history.
• Drug history and allergies.
• Family history.
• Social history: occupation, home circumstances, smoking tobacco and alcohol consumption.

The examination
• Preparation — see above:
  • position the patient flat with one pillow under the head
  • expose the abdomen from nipples to pubic symphysis but state that ideally you would like to expose the patient to mid-thigh.
• General examination from the end of the bed:
  • overall condition
  • drains, tubes, infusions, TPN, IDC, oxygen etc.
• Assessment for peripheral stigmata of chronic liver disease, unless instructed otherwise:
  • vital signs
  • hands: anaemia, clubbing, malnutrition, leukonychia, palmar erythema, bruising, asterixis (liver flap)
  • head and neck: cachexia (wasting of temporalis)/jaundice/pallor/lymphadenopathy, fetor hepaticus
  • chest: gynaecomastia, loss of body hair, spider naevi, bruising, pectoral muscle wasting, scratch marks, spider naevi.
• Examination of the abdomen:
  • check the surgical site for healing, infection, recurrence of hernia
  • hepatosplenomegaly, ascites, testicular atrophy, signs of portal hypertension, dilated abdominal wall veins (Caput medusae).
• Conclusion:
  • say that you would do a digital rectal examination
  • cover the patient
  • reposition the patient
  • thank the patient
  • clean your hands again
  • only now turn to face the examiners.

The discussion
• Relevant investigation of a patient with chronic liver disease:
  • FBC — look for anaemia, low platelet count, low WCC. Cirrhosis is often accompanied by anaemia, leukopenia and thrombocytopenia
  • coagulation studies — look for elevated INR, prolonged PT due to deficiency of the fat-soluble Vitamin K dependent clotting factors 2, 7, 9, 10 (the original terrestrial TV channels in Australia)
  • EUC — look for low Na, low K, metabolic alkalosis from recurrent vomiting, diarrhoea and hyperaldosteronism that accompanies cirrhosis
  • LFTs to differentiate cholestasis from hepatocellular injury and to assess level of hepatic decompensation
  • albumin — hypoalbuminaemia
  • hepatitis Serology — HCV, HBV
  • α-fetoprotein — HCC
  • liver biopsy — percutaneous liver biopsy is a useful in establishing the cause of cirrhosis and for assessing activity of the liver disease. Percutaneous liver biopsy is
not done when either coagulopathy or moderate ascites is present. In these situations, liver tissue can be obtained by means of a transjugular venous approach or laparoscopy.

- Assessment of functional hepatic reserve measured in chronic liver disease:
  - this can be measured by using the Child-Pugh or MELD scoring system. These scoring systems give a risk assessment of morbidity and mortality.

| Table 3.3.6 Child-Pugh criteria for chronic liver disease |
|---------------------------------|---|---|---|
| **MEASURE**                      | **1 POINT** | **2 POINTS** | **3 POINTS** |
| Encephalopathy (grade)           | None        | 1 or 2       | 3 or 4       |
| Ascites                          | None        | Mild         | Moderate     |
| Bilirubin (mg/dl)                | < 2.0       | 2.0–3.0      | ≥ 3.0        |
| Albumin (g/dl)                   | > 3.5       | 2.8–3.5      | < 2.8        |
| Prothrombin time prologation (s) (INR) | < 4 (< 1.7) | 4–6 (1.7–2.3) | > 6 (> 2.3) |

- Results in a score ranging from 5 to 15 points:
  - Class A = 5–6
  - Class B = 7–9
  - Class C = 10–15.
- Operative mortality risk according to Child-Pugh Class is:
  - A = 0–5% mortality
  - B = 10–15% mortality
  - C = >25% mortality.
- The overall 1-year survival rates for patients with Child-Pugh Class A, B, and C cirrhosis are approximately 100%, 80%, and 45% respectively.
- MELD score (Model of End stage Liver Disease), has been found to be as predictive of mortality as Child-Pugh. It is derived from Bilirubin + INR + Creatinine. It has been suggested that patients with a MELD score below 10 can undergo elective surgery, those with a MELD score of 10 to 15 may undergo elective surgery with caution, and those with a MELD score >15 should not undergo elective surgery.

- Assessment of the porto-systemic anatomy:
  - CT angiography is the test of choice for assessing the porto-systemic circulation and locating the presence of varices
  - Doppler ultrasonography is a non-invasive technique for assessment of portal venous patency, direction of portal flow, and shunt patency status
  - Ultrasound is also useful for assessing liver size, spleen size, and the presence of liver masses. It can also detect ascites in its earliest stages (≥100 mL)
  - Visceral angiography, but is not as popular as before because it is invasive.

- How do you treat an umbilical hernia in a patient with chronic liver disease?
  - It depends on the degree of liver disease and the nature of the presentation of the hernia and whether it is symptomatic and there is evidence of complication (strangulation/obstruction).
  - Patients with Child-Pugh class B and C should be managed conservatively unless there is a clear emergency surgical indication for surgery (e.g. strangulation/obstruction) due to the high risk of morbidity and mortality.
• Patients with asymptomatic hernias should be managed conservatively, with surgical correction of the hernia performed at the time of liver transplantation. The cornerstone of conservative management in asymptomatic patients with umbilical hernias is aggressive management of ascites. Elastic/velcro abdominal binders can also help reduce pain and minimise further enlargement of the hernia.
• All patients should be managed in a multidisciplinary team environment (hepatologist, surgeons, nursing and allied health staff capable of looking after such patients).
• Patients should be optimised medically if surgery is being contemplated with:
  • reduction in ascites (salt restriction, spironolactone ± frusemide)
  • nutritional optimisation (high protein high calorie supplements)
  • treatment of portal hypertension (propanolol/TIPPS).

3.3.7 Liver metastases

The history
• Introduce yourself and ask the patient what has been going on. The patient may indicate that he had bowel cancer that spread to the liver.
• Try to maintain a structure to your history, using the framework above. Begin by orientating yourself.
  • Has the liver metastasis been removed?
  • Has the bowel cancer been removed?
  • Was the liver metastasis removed before or after the bowel cancer?
• The detail relating to the bowel cancer:
  • Was the spread to his liver discovered at the same time as the bowel cancer or afterwards?
  • Did he have any treatment before the bowel surgery (particularly chemotherapy)?
• What bowel surgery did he have done? Laparoscopic surgery (keyhole) or open? What bowel operation? Patients will usually know if the left or right or most of the colon was removed.
• Did he have a stoma or peri-operative complication?
• Did he have chemotherapy post-operatively?
• What follow-up imaging/colonoscopy has he had?
• The detail relating to the liver metastasis:
  • When were the liver metastasis discovered?
  • How were they picked up, on a CT scan or PET or MRI?
  • Has he had surgery yet?
  • If he has had surgery what type?
  • Was it a major liver resection?
  • Did he require pre-operative portal vein embolisation?
  • Did he have a post-operative complication such as bile leak or return to theatre?
• Current functional status (diet, mobility, bowel function, expected date of discharge).
• Risk factor assessment:
  • personal or family history of polyps/colorectal cancer/inflammatory bowel disease.
• Past medical history.
• Surgical history.
• Drug history and allergies.
• Family history — particularly of endometrial/gastric/ovary/urothelium (HNPCC).
• Social history: occupation, home circumstances, smoking tobacco and alcohol consumption.

The examination
• Preparation (see above):
  • position the patient flat with one pillow under the head
  • expose the abdomen from nipples to pubic symphysis but state that ideally you would like to expose the patient to mid-thigh.
• General examination from the end of the bed:
  • overall condition
  • if post-operative: drains, tubes, infusions, TPN, IDC, oxygen etc.
• Assessment for peripheral signs, unless instructed otherwise:
  • vital signs.
• Examination of the abdomen:
  • inspection: look for previous scars and presence of a portacath — get the patient to point out scars. (Note: Unless the patient has had laparoscopic liver surgery they will have a subcotal or upper midline incision. There may be a diverting ileostomy from an ultra-low rectal resection. The colonic surgery is often laparoscopic and so the incisions may be hard to see. Look for a portacath.). Inspect drains, dressings, bruising. Inspect wounds for signs of infection
  • palpation: soft; tender; organomegaly (liver edge); ascites. Assess groin and incision for evidence of a cough impulse. Palpate the testicles
  • percussion: not usually relevant post-operatively unless distended and you want to determine if it is gaseous or fluid (ascites/ileus). Percussion for shifting dullness; fluid thrill
  • auscultation: listen in four quadrants for the character of borborygmi, bruits over the renal arteries, aorta and liver.
3.3 Common medium cases

- Conclusion:
  - say that you would do a digital rectal examination in the pre-operative setting
  - cover the patient
  - reposition the patient
  - thank the patient
  - clean your hands again
  - only now turn to face the examiners.

The discussion
- The examiners may ask you to tell them what you found. An example of a succinct summary for this case would be as follows. ‘Mr Y is a 59-year-old man who had a right hemicolectomy 3 years ago for a colonic cancer. He received adjuvant chemotherapy after his colonic resection. Six months ago he was discovered to have a liver metastasis because of a rising CEA noted by his GP. The tumour was in the right side of the liver and was large. He had what sounds like a portal vein embolisation and then an extended hepatectomy. He was in hospital for 4 weeks after his liver surgery. It sounds like he may have had a bile leak and liver failure post-operatively, but he made a complete recovery.’
- Detection and assessment of liver metastases after surgery for colorectal cancer
  - All patients who have undergone curative surgery and are fit for further intervention should be offered surveillance to detect metachronous colorectal cancers and liver metastases.
  - This follow-up involves CEA measurement, CT scanning of the chest/abdomen/pelvis and colonoscopy; the former two investigations are integral to the early detection of (liver) metastases.
  - CEA measurement and CT scanning is usually arranged 3–6 monthly in conjunction with the clinical review of the patient. There is evidence of survival benefit to this practice when compared with no surveillance at all. RCTs comparing intensive (including CEA/CT) vs less intensive surveillance show an overall survival benefit to intensive follow-up. Follow-up should be 3–6 monthly for 2 years and 6 monthly thereafter.
  - Once a liver metastasis is diagnosed, search for extra-hepatic disease using PET should be considered in high-risk patients. PET will detect metastasis occult to other staging modalities and change clinical management in about 25% of cases. However, after commencement of chemotherapy, PET has a significant false-negative rate because of altered metabolic activity within the lesions.
  - Non-therapeutic laparotomy can be avoided using laparoscopy and intra-operative US. This allows the evaluation of visceral and parietal surfaces of the abdomen (and biopsy of suspicious lesions), the porta hepatitis for occult nodal disease. Intra-operative US can be used to detect unexpected lesions in the ‘future’ liver remnant.
  - Portal hypertension can also be evaluated and the normal liver biopsied.
  - The Clinical Risk Score (CRS) can be used to predict which patients are most likely to be non-resectable at laparoscopy.
  - Within the CRS a point is given for each of:
    - > 1 liver tumour
    - liver tumour >5 cm
    - node-positive colorectal primary
    - disease free interval <1 yr and
    - CEA >200.
• If the score is 0, patients are likely to remain resectable at laparoscopy. If the score is 4–5, 25% of patients will be non-resectable at laparoscopy. Laparoscopy should be restricted to those with high CRS.

• What are the principles of treatment of liver metastases after surgery for colorectal cancer?

• About 20% of patients have liver metastasis at initial presentation of a colorectal cancer (synchronous).

• About 30% of patients develop metastases after resection of a colorectal cancer (metachronous).

• Approximately one-quarter of the liver metastasis are resectable, although liver surgeons continue to push the boundaries with safe liver resection.

• In appropriately selected patients, liver resection offers 5-year relapse-free survival rates of 30% and 5-year overall survival rates of approximately 58%.

• The optimal selection of patients for resection continues to evolve. However, resection should not be considered in the following situations:
  • non-resectable extrahepatic disease detected by CT, PET scans or laparoscopy
  • involvement of the CHA or main portal vein
  • inadequate liver remnant after resection
  • unfit for surgery.

• Modern multidisciplinary consensus defines resectable CRC liver metastases as tumours that can be resected completely, leaving an adequate liver remnant.

• The liver remnant must retain adequate hepatic arterial and portal venous inflow and venous and biliary drainage. Future liver remnant (FLR) is calculated by CT volumetric study. The normal liver constitutes approximately 2% of body weight. For a successful liver resection FLR needs to be:
  • normal liver: 20% or > if pre-operative chemo
  • cirrhosis/diabetics: >40%.

• Consider portal vein embolisation if:
  • FLR <20% in normal patients or
  • FLR <40% in cirrhotics/diabetics.

• What are the main approaches for the treatment of resectable liver metastases after surgery for colorectal cancer?

• Liver first approach:
  • this option is usually favoured if the primary is under control (non-obstructing/no bleeding) and there is large volume liver disease. Most liver surgeons believe that the liver is the determining factor for survival and therefore argue that the liver resection should be done first. Also, in the case of rectal cancer, liver surgeons argue that a complication post-pelvic surgery can delay liver surgery to the point where the patient may become inoperable.

• Synchronous resection approach:
  • this approach should be considered for patients with low volume uncomplicated liver resection; for example, single solitary peripheral metastasis or segment two-thirds liver resection where there is a lesser risk of peri-operative and post-operative morbidity and mortality.

• Primary first approach:
  • this is the preferred pathway in most institutions, as the primary tumour is believed to be the source of the metastatic cells. Liver resection occurs 6–8/52 after bowel resection.

• Chemotherapy first approach:
  • chemotherapy/re-evaluate/± liver resection. This approach is preferred in patients with high volume liver disease, as if there is disease progression on
chemotherapy then resection is not suitable. This can spare the patient liver resection. If, on the other hand, the disease has responded or is stable, resection of the metastatic disease should be attempted. No more than two to three courses of pre-operative chemotherapy are recommended before liver resection as it causes CASH (chemotherapy-associated steatohepatitis). The approach to treatment is not standardised and varies between institutions and countries. The optimal chemotherapy regimen is not established, but FOLFOX/ FOLFIRI are considered to be reasonable choices. For non-resectable disease, ‘up front’ chemotherapy is an appropriate option in an attempt to down-staging a patient to the point of resectability. This occurs in approximately 10–15% of patients. Note, longer durations of pre-operative chemotherapy increase the potential for liver toxicity and post-operative complications.

- Non-resectable liver metastases should be considered for RFA/TACE.
- Following complete resection of liver metastases, the best post-operative strategy is uncertain. In the absence of published randomised trials to guide clinical practice following metastasectomy, completion of a full 6 month course of systemic chemotherapy containing oxaliplatin is recommended.

### 3.3.8 Abdominal mass

You are asked to take a history and examine a patient who has presented with an abdominal mass. Outlined below are the pertinent aspects of history, examination and presentation of the differential diagnosis and treatment plan.

Consider the classification of abdominal masses. They can be classified by location, region or aetiology.

- **Location** — a good idea to keep this classification in mind during the history and examination:
  - anterior abdominal wall
  - intraperitoneal
  - retroperitoneal.

- **Region**:
  - right upper/lower quadrant
  - left upper/lower quadrant
  - midline masses may be supraumbilical or infraumbilical.

- **Aetiology**:
  - neoplastic (benign or malignant)
  - infective (e.g. hydatid cyst, liver abscess)
  - inflammatory (e.g. phlegmon secondary to appendicitis or Crohn’s disease)
  - vascular (aneurysm, rectus sheath haematoma).

#### The history

- Introduce yourself and ask the patient what has been going on.
- Try to maintain a structure to your history, using the framework above. Begin by orientating yourself (see above).
- Be aware that the lump may have been an incidental finding and not associated with any clinical symptoms. Even if this is the case you should run through the list of questions below to demonstrate to the examiners that you know what you should ask about.
- The presenting symptoms and history of presenting complaint:
  - **the lump**: when first appeared, how noticed, progression since then (bigger?), any other lumps
• associated symptoms: pain (SOCRATES) or distension
• GI symptoms (from upper to lower GI tract): dysphagia, dyspepsia, early satiety, postprandial vomiting, reflux, haematemesis, jaundice, abdominal distension, altered bowel habit (constipation/ diarrhoea/ both)
• other associated symptoms — systemic: fevers, weight loss; anorexia; malaise; shortness of breath
• other associated symptoms — urinary: frequency, dysuria, haematuria, pneumaturia, UTI
• other associated symptoms — gynaecological in females: menstrual cycle, menorrhagia, dysmenorrhoea, intermenstrual bleeding, last Pap smear, obstetric history
• other associated symptoms — vascular: claudication, risk factors for cardiovascular disease, aneurysms
• investigations:
  • details of those performed to date
  • results
  • presumed diagnosis
• treatment:
  • Was the patient admitted electively or acutely?
  • What procedure was performed?
  • Was neo-adjuvant treatment required or discussed?
  • Were there any complications after the operation?
  • Current status of the patient (diet, mobility, bowel function, expected date of discharge).
  • Will adjuvant treatment be required (radiotherapy or chemotherapy)?
• Risk factor assessment:
  • personal or family history of polyps/colorectal cancer/inflammatory bowel disease.
• Past medical history:
  • history of malignancy (including GIT, breast and melanoma) and/or prior surgery including non-abdominal surgery (including skin excisions)
  • previous gastroscopy/colonoscopy (when/where/findings)
  • pancreatitis
  • IBD.
• Surgical history.
• Drug history and allergies.
• Family history — history of malignancy within family members and other diseases within the family including AAA.
• Social history: occupation, home circumstances, smoking tobacco and alcohol consumption, travel/migration.

The examination
• Preparation (see above):
  • position the patient flat with one pillow under the head
  • expose the abdomen from nipples to pubic symphysis but state that ideally you would like to expose the patient to mid-thigh.
• General examination from the end of the bed:
  • overall condition
  • drains, tubes, infusions, TPN, IDC, oxygen etc if post-operative.
• Assessment for peripheral signs, unless instructed otherwise:
  • vital signs
• hands: stigmata of gastrointestinal disease — Dupuytren’s contractures, nail changes (leukonychia, clubbing), pallor of the palmer creases (anaemia). Ask the patient to squeeze your hand as poor grip strength is a marker of inadequate nutrition

• examine the head and neck: cachexia (wasting of temporalis assess the eyes looking at the sclera for icterus and the conjunctiva for pallor; assess the mouth for angular stomatitis and glossitis

• examine the supraclavicular fossa for nodal metastases

• examine the chest for gynaecomastia, skin changes, auscultate for pleural effusions.

• Examination of the abdomen:
  • inspection: careful inspection as often the mass will be obvious by kneeling beside the patient with your eyes level with the abdomen. Observe movement with respiration; distension; scars; stomas; dressings; bruising. Inspect wounds for signs of infection. Look at drains, stomas and urine bag if present
  • palpation: soft; tender; organomegaly; ascites
  • percussion: not usually relevant post-operatively unless distended and you want to determine if it is gaseous or fluid (ascites/ileus). Percussion for shifting dullness; fluid thrill
  • auscultation: listen in four quadrants for the character of borborygmi, bruits over the renal arteries, aorta and liver:
    • for any mass it is important to describe it according to the mnemonic ‘Should The Children Ever Find Lumps Readily’ (see below, and Chapter 3.4, exam technique). In addition, it is crucial to assess the following to help distinguish the aetiology of an abdominal mass. Use the acronym SPRUE as an aide-memoire:
      • site of enlargement
      • percussion note
      • respiratory movement
      • unable to get above it
      • edge characteristics.

• Conclusion:
  • say that you would do a digital rectal examination in the pre-operative setting.
  • cover the patient
  • reposition the patient
  • thank the patient
  • clean your hands again
  • only now turn to face the examiners.

The discussion

• Having completed a history and examination the differential diagnosis needs to be considered. Refer to Chapter 3.5, Figure 3.5.13 and Table 3.3.8A, for a full list of differentials. Differential diagnosis of causes of intra-abdominal masses by location and organ of origin in the abdomen.

• Present a list of the differential diagnoses that you consider are most likely in the patient you have seen. They could be ordered from most common to least common or you could start with those that are most important to exclude (e.g. malignancy).
• Present a management strategy for the patient. Your management should include whether the patient needs admission to hospital or can be managed as an outpatient (i.e. is the patient sick?). Further investigation involves blood tests, imaging and endoscopy as appropriate to the patient.

  - **Blood tests:**
    - FBC — anaemia, leucocytosis
    - coagulation profile — INR raised in liver synthetic dysfunction, pre-operative
    - EUC — electrolytes, renal impairment
    - LFTs — hepatic dysfunction, raised bilirubin
    - amylase/lipase if indicated
    - tumour markers if indicated (CEA — colon; Ca19.9 — pancreas, gastric, biliary; AFP — HCC, testicular; Ca125 — ovarian)
    - HBV/HCV/Hydatid/amoebic serology if indicated.

  - **Imaging:**
    - a CT abdomen/pelvis with iv and oral contrast is the best initial test for an undifferentiated abdominal mass
    - other imaging modalities that may be useful — vascular duplex ultrasound (AAA); pelvic ultrasound
    - CXR should be obtained routinely if surgery is considered and as a screening test for thoracic pathology
    - in certain instances further imaging may be required (e.g. 4 phase CT scan for characterisation of liver or pancreatic tumours or MRI).

  - **Endoscopy:**
    - upper GI endoscopy if the origin is thought to be UGIT
    - ERCP may be indicated in the setting of a RUQ mass and biliary obstruction
    - endoscopic ultrasound and FNA is valuable in the assessment of pancreatic masses
    - colonoscopy if suspected colonic malignancy to confirm diagnosis and exclude synchronous lesions.

- After presenting the differential diagnoses and a management plan, list the issues that need to be addressed. The list may include:
  - confirmation of diagnosis
  - staging in setting of malignancy
  - MDT discussion of patients with malignancies with consideration to surgical or non-surgical management and any indications for neo-adjuvant or adjuvant treatment
  - management of sepsis (if present)
  - disease specific management or appropriate referral
  - nutrition
  - immediate and longer term treatment goals.

- The clinical profile, examination, investigations, aetiology and discussion points for intra-abdominal masses are detailed in Tables 3.3.8A, B, C and D.

| Table 3.3.8A Clinical profile, examination, investigations, aetiology and discussion points for intra-abdominal masses (splenomegaly, gall bladder mass, liver mass) |
|---------------------------------|---------------------------------|---------------------------------|---------------------------------|
| **Splenomegaly** | **Gall Bladder Mass** | **Liver Mass** |
| Mass symptoms | Asymptomatic | Asymptomatic | Asymptomatic |
| | Progressive enlargement | Acute hx if infective cause | Progressive enlargement |
| | | Chronic hx if malignant | Generalised if ascites |
### Table 3.3.8A  Continued

<table>
<thead>
<tr>
<th></th>
<th>SPLENOMEGALY</th>
<th>GALL BLADDER MASS</th>
<th>LIVER MASS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Painful mass</strong></td>
<td>Discomfort or acute pain if infarction or infection Can radiate to left shoulder</td>
<td>Yes (infective) Radiates back, epigastrium or right shoulder</td>
<td>Pain associated with acute causes, bleeding from lesion, congestion</td>
</tr>
<tr>
<td><strong>Appetite</strong></td>
<td>Can be reduced</td>
<td>Reduced</td>
<td>Can be reduced</td>
</tr>
<tr>
<td><strong>Nausea/vomiting</strong></td>
<td>No</td>
<td>Yes (infection, jaundice)</td>
<td>Not usually</td>
</tr>
<tr>
<td><strong>Haematemesis</strong></td>
<td>Low platelets or portal HT</td>
<td>No</td>
<td>Low platelets or portal HT</td>
</tr>
<tr>
<td><strong>Fullness/bloating</strong></td>
<td>Yes (massive spleen)</td>
<td>No</td>
<td>Some liver conditions</td>
</tr>
<tr>
<td><strong>Dyspepsia</strong></td>
<td>Yes (massive spleen)</td>
<td>Can occur with gall stones</td>
<td>Some liver conditions</td>
</tr>
<tr>
<td><strong>Weight loss</strong></td>
<td>Yes</td>
<td>Can occur with malignancy</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Altered bowel habit</strong></td>
<td>No</td>
<td>No</td>
<td>No unless colorectal cancer metastases</td>
</tr>
<tr>
<td><strong>Jaundice etc</strong></td>
<td>Assoc with liver disease, haemolytic anaemia</td>
<td>Yes — stones, malignancy</td>
<td>Yes</td>
</tr>
<tr>
<td><strong>Fever etc</strong></td>
<td>Yes</td>
<td>Yes — infection</td>
<td>Can occur</td>
</tr>
<tr>
<td><strong>Past history</strong></td>
<td>Immunological disorders Abnormal FBC Hepatitis Risk factors (RF) for hepatitis/chronic liver disease (IVDU/ alcohol) FHx anaemia, RA, SLE</td>
<td>Known gall stones Previous cholecystitis</td>
<td>Malignancy Hepatitis and RF for hepatitis/chronic liver disease (IVDU/ alcohol) Haematological disorders Cardiovascular disease FHx: polycystic disease</td>
</tr>
<tr>
<td><strong>Examination</strong></td>
<td>Spleen enlarges inferomedially; moves down with respiration Splenic notch Bimanual palpation Signs rheumatological disease, chronic liver disease, lymphadenopathy</td>
<td>Tender poorly defined mass that cannot get above in the midclavicular line on the right (infective causes) Non tender mass associated with jaundice in this location suggests malignancy of distal CBD</td>
<td>Tender diffuse liver enlargement with acute infective hepatitis Non-tender hard, irregular liver indicates malignancy Cystic lesions — smooth Signs of chronic liver and rheumatological disease</td>
</tr>
<tr>
<td><strong>Investigations</strong></td>
<td>FBC, LFTS, EUC, CRP</td>
<td>FBC, LFTS, EUC, CRP</td>
<td>FBC, LFTS, EUC, CRP</td>
</tr>
<tr>
<td>Blood smear examination</td>
<td>Coagulation studies</td>
<td>Coagulation studies</td>
<td></td>
</tr>
<tr>
<td>Coagulation studies</td>
<td>CA 19.9, CEA</td>
<td>CA 19.9, CEA, AFP, CA 15.3</td>
<td></td>
</tr>
<tr>
<td>Blood cultures</td>
<td>Ultrasound</td>
<td>Ultrasound (cystic vs solid)</td>
<td></td>
</tr>
<tr>
<td>Viral serology (EBV, CMV) Echinococcus serology</td>
<td>CT Abdomen/Pelvis</td>
<td>Viral serology (EBV, CMV) Echinococcus, hepatitis</td>
<td></td>
</tr>
<tr>
<td>CT Abdomen/Pelvis</td>
<td>Blood cultures</td>
<td>CT Abdomen/Pelvis</td>
<td></td>
</tr>
<tr>
<td>CT Chest/neck</td>
<td>EUS</td>
<td>PET scan</td>
<td></td>
</tr>
<tr>
<td>PET scan</td>
<td>ERCP/MRCP</td>
<td>MRI liver</td>
<td></td>
</tr>
<tr>
<td>Bone marrow bx</td>
<td>Gastroscopy/colonoscopy</td>
<td>(GIT primary)</td>
<td></td>
</tr>
</tbody>
</table>
### Table 3.3.8A Continued

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>SPLENOMEgALY</th>
<th>GALL BLADDER MASS</th>
<th>LIVER MASS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haematological malignancy</td>
<td>Mucocele</td>
<td>HCC, cholangiocarcinoma</td>
<td></td>
</tr>
<tr>
<td>Myeloproliferative disease</td>
<td>Acute cholecystitis</td>
<td>Metastases (colorectal)</td>
<td></td>
</tr>
<tr>
<td>Infections (EBV, CMV, malaria, infective endocarditis)</td>
<td>Empyema</td>
<td>Infections (acute hepatitis, EBV, CMV, hydatid cyst)</td>
<td></td>
</tr>
<tr>
<td>Portal hypertension</td>
<td>Biliary obstruction</td>
<td>Hepatic adenoma, FNH</td>
<td></td>
</tr>
<tr>
<td>Immunological (RA, SLE)</td>
<td>Gall bladder carcinoma</td>
<td>Polycystic disease</td>
<td></td>
</tr>
<tr>
<td>Infiltrative (e.g. sarcoidosis)</td>
<td></td>
<td>Right heart failure</td>
<td></td>
</tr>
<tr>
<td>Splenic cysts</td>
<td></td>
<td>Haematological diseases</td>
<td></td>
</tr>
<tr>
<td>Metastases (lung, pancreas, stomach)</td>
<td></td>
<td>Riedels lobe</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Simple cyst, cystadenoma</td>
<td></td>
</tr>
</tbody>
</table>

### Discussion points

- **Indications for splenectomy**
- **Management of obstructive jaundice**
- **Management of hepatic colorectal metastases**
- **Post-operative complications of splenectomy**
- **Management of gall bladder carcinoma**
- **Management of HCC**
- **OPSI and its prevention**
- **Management of acute cholecystitis**
- **Management of hydatid disease**

### Table 3.3.8B Clinical profile, examination, investigations, aetiology and discussion points for intra-abdominal masses (pancreas, stomach, colon)

<table>
<thead>
<tr>
<th>PANCREAS</th>
<th>STOMACH</th>
<th>COLON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass symptoms</td>
<td>Progression over time</td>
<td>Progression over time</td>
</tr>
<tr>
<td>Painful mass</td>
<td>Yes — radiating to back</td>
<td>Often non-tender</td>
</tr>
<tr>
<td>Appetite</td>
<td>Reduced</td>
<td>Reduced</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Haematemesis</td>
<td>Can occur if tumour erodes into duodenum/stomach.</td>
<td>Can be present</td>
</tr>
<tr>
<td>Fullness/bloating</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Dyspepsia</td>
<td>Can occur</td>
<td>Present</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Altered bowel habit</td>
<td>Neuroendocrine tumours can cause diarrhoea</td>
<td>No</td>
</tr>
<tr>
<td>Jaundice etc</td>
<td>Can occur</td>
<td>No</td>
</tr>
</tbody>
</table>
### Table 3.3.8B continued

<table>
<thead>
<tr>
<th>PANCREAS</th>
<th>STOMACH</th>
<th>COLON</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fevers etc</td>
<td>Can occur</td>
<td>No</td>
</tr>
<tr>
<td>Urinary symptoms</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Past history</td>
<td>Pancreatitis, Diabetes, Alcoholism</td>
<td>Peptic ulcer/H. pylori, Previous gastrectomy/ulcer surgery, FHx: stomach cancer</td>
</tr>
<tr>
<td>Examination</td>
<td>Carcinoma: cachexia and a firm poorly defined fixed mass deep in epigastrium, Pseudocyst: smooth poorly defined epigastic mass</td>
<td>Cachexia, Usually a poorly defined mass in epigastrium, Gastric distension can be present (succussion splash)</td>
</tr>
<tr>
<td>Investigations</td>
<td>FBC, LFTS, EUC, lipase, CRP</td>
<td>FBC, LFTS, EUC, CRP</td>
</tr>
<tr>
<td>Coagulation studies</td>
<td>CEA, CA 72.4, CA 19.9</td>
<td>CEA</td>
</tr>
<tr>
<td>CA 19.9, CEA</td>
<td>Endoscopy + biopsy</td>
<td>Colonoscopy + bx</td>
</tr>
<tr>
<td>EUS/ERCP/MRCP</td>
<td>CT Chest/Abdomen/Pelvis</td>
<td>Blood cultures, MSU</td>
</tr>
<tr>
<td>CT Chest/Abdomen/Pelvis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aetiology</td>
<td>Carcinoma pancreas</td>
<td>Carcinoma stomach</td>
</tr>
<tr>
<td>Pancreatic pseudocyst</td>
<td>GIST stomach</td>
<td>Diverticular abscess/phlegmon/fistula</td>
</tr>
<tr>
<td>Neuroendocrine tumour</td>
<td>Neuroendocrine tumour</td>
<td></td>
</tr>
<tr>
<td>Discussion points</td>
<td>Management of pancreatic pseudocysts</td>
<td>Management of adenocarcinoma of stomach</td>
</tr>
<tr>
<td>Management of pancreatic cancer</td>
<td>Management of GIST tumours</td>
<td>Management of diverticular abscess</td>
</tr>
</tbody>
</table>

### Table 3.3.8C Clinical profile, examination, investigations, aetiology and discussion points for intra-abdominal masses (omentum, ovarian, uterine)

<table>
<thead>
<tr>
<th>OMENTAL MASS</th>
<th>OVARIAN MASS</th>
<th>UTERINE MASS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mass symptoms</td>
<td>Generalised distension if ascites present</td>
<td>Acute onset if due to ectopic/torsion</td>
</tr>
<tr>
<td>Painful mass</td>
<td>Discomfort more common</td>
<td>Ectopic, torsion, endometrioma associated with acute pain</td>
</tr>
<tr>
<td>Appetite</td>
<td>Reduced</td>
<td>Usually normal</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>Can be present</td>
<td>Can occur with acute pathologies</td>
</tr>
<tr>
<td>Haematemesis</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Fullness/bloating</td>
<td>Yes</td>
<td>Associated with malignancy</td>
</tr>
<tr>
<td>Examination Surgery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------------</td>
<td></td>
<td></td>
</tr>
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### Table 3.3.8C Continued

<table>
<thead>
<tr>
<th>Symptom / Examination / Investigations / Aetiology / Discussion points</th>
<th>OMENTAL MASS</th>
<th>OVARIAN MASS</th>
<th>UTERINE MASS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspepsia</td>
<td>Can be present</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Yes</td>
<td>Yes if malignancy</td>
<td>Yes if malignancy</td>
</tr>
<tr>
<td>Altered bowel habit</td>
<td>Constipation</td>
<td>No</td>
<td>Constipation</td>
</tr>
<tr>
<td>Jaundice etc</td>
<td>Can be present</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Fevers etc</td>
<td>Often absent</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Urinary symptoms</td>
<td>Bladder compression can cause frequency</td>
<td>Bladder compression can cause frequency</td>
<td>Bladder compression and fistulae can cause irritative symptoms</td>
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<tr>
<td>Gynaecological symptoms</td>
<td>If associated with gynaecological primary</td>
<td>Symptoms associated with menstrual cycle</td>
<td>Symptoms associated with menstrual cycle</td>
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<tr>
<td></td>
<td></td>
<td>Possible pregnancy</td>
<td>Possible pregnancy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dysfunctional uterine bleeding</td>
<td>Dysfunctional uterine bleeding</td>
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<tr>
<td></td>
<td></td>
<td>Menopause</td>
<td>Menopause</td>
</tr>
<tr>
<td>Past history</td>
<td>Malignancy</td>
<td>Malignancy (breast)</td>
<td>Malignancy (breast)</td>
</tr>
<tr>
<td></td>
<td>Appendicectomy</td>
<td>Parity</td>
<td>Parity</td>
</tr>
<tr>
<td></td>
<td>Ovarian pathology</td>
<td>PCOS</td>
<td>OCP/HRT</td>
</tr>
<tr>
<td></td>
<td>FHx: malignancy (colorectal, gynaecological)</td>
<td>Endometriosis</td>
<td>FHx: malignancy (breast, gynaecological)</td>
</tr>
<tr>
<td>Examination</td>
<td>Cachexia</td>
<td>Ovarian masses can be felt deep in iliac fossae but are better assessed with bimanual palpation</td>
<td>Uterine masses can be felt suprapubically Unable to get below PV and bimanual examination</td>
</tr>
<tr>
<td></td>
<td>Ascites</td>
<td>Determine fixation</td>
<td>PR</td>
</tr>
<tr>
<td></td>
<td>Often multiple small hard non-tender masses with fixation to anterior abdominal wall</td>
<td>Examine cervix</td>
<td>PR — Blummers shelf</td>
</tr>
<tr>
<td></td>
<td>PR — Blummers shelf</td>
<td></td>
<td></td>
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<tr>
<td>Investigations</td>
<td>FBC, EUC, LFT, CRP</td>
<td>FBC, EUC, LFT, CRP</td>
<td>FBC, EUC, LFT, CRP</td>
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<td>CA 125, CEA, CA 72.4, CA 19.9</td>
<td>CA 125, AFP, β-HCG</td>
<td>CA 125, CA</td>
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<tr>
<td></td>
<td>CT abdomen/pelvis</td>
<td>Pelvic ultrasound</td>
<td>Pelvic ultrasound</td>
</tr>
<tr>
<td></td>
<td>Ascitic tap for cytology</td>
<td>CT abdomen/pelvis</td>
<td>CT abdomen/pelvis</td>
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<tr>
<td></td>
<td>Gastroscopy/colonoscopy</td>
<td>Diagnostic laparoscopy</td>
<td>Hysteroscopy + bx</td>
</tr>
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<tr>
<td>Aetiology</td>
<td>Peritoneal mesothelioma</td>
<td>Malignant tumour</td>
<td>Carcinoma</td>
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<tr>
<td></td>
<td>Pseudomyxoma peritonei</td>
<td>Torsion</td>
<td>Fibroids</td>
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<tr>
<td></td>
<td>Metastases — ovarian, stomach, pancreas, colorectal</td>
<td>Cysts</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ectopic pregnancy</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dermoid</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Endometrioma</td>
<td></td>
</tr>
<tr>
<td>Discussion points</td>
<td>Pathophysiology of pseudomyxoma peritonei</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Management of unknown primary with omental metastases</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>RENAL MASS</td>
<td>BLADDER MASS</td>
<td>SMALL INTESTINE</td>
</tr>
<tr>
<td>-------------------------</td>
<td>----------------------------------</td>
<td>------------------------------------</td>
<td>--------------------------------------</td>
</tr>
<tr>
<td>Mass symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Painful mass</td>
<td>Back or loin pain</td>
<td>Suprapubic pain, flank pain due to ureteric obstruction</td>
<td>Periumbilical discomfort or colicky pain if SBO</td>
</tr>
<tr>
<td>Appetite</td>
<td>Usually normal</td>
<td>Usually normal</td>
<td>Can be reduced</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Haematemesis</td>
<td>No</td>
<td>No</td>
<td>Usually no</td>
</tr>
<tr>
<td>Fullness/bloating</td>
<td>No</td>
<td>Suprapubic swelling</td>
<td>Yes</td>
</tr>
<tr>
<td>Dyspepsia</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Yes if malignancy</td>
<td>Yes if malignancy</td>
<td>Yes if malignancy</td>
</tr>
<tr>
<td>Altered bowel habit</td>
<td>No</td>
<td>No</td>
<td>Yes — constipation, diarrhoea (carcinoid)</td>
</tr>
<tr>
<td>Jaundice etc</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Fevers etc</td>
<td>Yes (paraneoplastic)</td>
<td>No</td>
<td>Yes — carcinoid</td>
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<tr>
<td>Urinary symptoms</td>
<td>Haematuria</td>
<td>Irritative symptoms or obstructive</td>
<td>No</td>
</tr>
<tr>
<td>Past history</td>
<td>Occupation — carcinogen exposure</td>
<td>Occupation — carcinogen exposure</td>
<td>Malignancy IBD or FHX IBD Abdominal surgery</td>
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<tr>
<td></td>
<td>Smoking</td>
<td>Smoking</td>
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<td>Obesity</td>
<td>Recurrent UTI</td>
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<td>Analgesic abuse</td>
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<tr>
<td></td>
<td>HTN, DM</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>FHx: polycystic disease, renal failure</td>
<td></td>
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</tr>
<tr>
<td>Examination</td>
<td>Cachexia</td>
<td>Distended fluid filled mass that you cannot get below PR to examine prostate</td>
<td>Generalised abdominal distension (SBO) Discrete mass uncommon Examine hernia orifices and for incisional hernia</td>
</tr>
<tr>
<td></td>
<td>Flank mass that may be balloted</td>
<td></td>
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<tr>
<td></td>
<td>Scrotal varices</td>
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<td>Polycystic liver</td>
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<td>Investigations</td>
<td>FBC, EUC, LFTs, CMP</td>
<td>FBC, EUC, LFTs, CMP</td>
<td>FBC, EUC, LFTs, CMP</td>
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<td>MSU</td>
<td>PSA</td>
<td>Chromogranin A</td>
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<td>Renal tract ultrasound</td>
<td>MSU</td>
<td>CT abdomen/pelvis</td>
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<td>CT Abdomen/Pelvis</td>
<td>CT abdomen/pelvis/IVP</td>
<td>Octreotide SPECT/CT</td>
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<td>Urine cytology</td>
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<td>Colonoscopy with TI intubation</td>
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<td>Cystoscopy + bx</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TRUS prostate + bx</td>
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### Table 3.3.8D Continued

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>RENAL MASS</th>
<th>BLADDER MASS</th>
<th>SMALL INTESTINE</th>
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<tbody>
<tr>
<td>Carcinoma</td>
<td></td>
<td>Bladder neck obstruction — prostate cancer/BPH</td>
<td>Adenocarcinoma</td>
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<tr>
<td>Polycystic disease/simple cysts</td>
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<td>Bladder tumour</td>
<td>GIST</td>
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<tr>
<td>Hydronephrosis</td>
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<td>Carcinoid tumour</td>
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<tr>
<td>Pyonephrosis</td>
<td></td>
<td></td>
<td>Lymphoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Small bowel obstruction</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Terminal ileitis</td>
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</tbody>
</table>

**Discussion points**

<table>
<thead>
<tr>
<th></th>
<th>Management of carcinoid tumour and carcinoid syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Management of SBO</td>
</tr>
</tbody>
</table>

### 3.3.9 Malignant melanoma

The possible scenarios that you will encounter include a patient with either:
- stage I/II disease: localised melanoma, further investigation and treatment depends on pathology of primary lesion
- stage III disease: nodal metastases. Includes nodal recurrence and in-transit disease, or
- stage IV disease: systemic metastases.

**The history**

- Introduce yourself and ask the patient what has been going on. The patient may indicate that he had an operation for rectal cancer last week and now has a stoma bag for 3 to 6 months.
- Try to maintain a structure to your history, using the framework above. Begin by orientating yourself.
- The presenting symptoms and history of the primary melanoma:
  - suspicious features (ABCDE of melanoma)
  - excision or partial/punch biopsy
  - pathology of the melanoma — Breslow, ulceration, mitoses, margins.
- History of lymph node(s) assessment:
  - Is there a palpable nodal mass/lymphoedema?
  - Was sentinel lymph node biopsy (SLNB) performed?
  - What were the number of nodes removed, presence of metastases?
  - Lymph node (LN) dissection — for clinically evident disease or positive SLN or for nodal relapse?
  - Were there complications from any surgery?
- History of systemic metastases:
  - where, how many, symptomatic versus non-symptomatic
  - weight loss, anaemia, neurological symptoms
• history of resection versus non-operative management (e.g. Radiotherapy [RTx], BRAFi, chemotherapy).

• Adjuvant treatment:
  • RTx, IFN, chemotherapy, ipilimumab, BRAF inhibitors, trials, (isolated limb infusion) ILI.

• Risk factor assessment:
  • personal or family history of previous melanoma/non-melanoma skin cancers
  • history of sun exposure (e.g. occupation).

• Past medical history.

• Surgical history.

• Drug history and allergies.

• Family history.

• Social history: home circumstances, smoking tobacco and alcohol consumption.

The examination

• Preparation (see above):
  • position the patient comfortably
  • expose the patient completely, while preserving his/her dignity as much as possible.

• Inspection:
  • comment on general appearance and then general features of skin damage (e.g. fair skin with solar keratoses/BCC/SCC/multiple previous excision sites)
  • look for evidence of RTx changes
  • evidence of recent/previous surgery: where, drains and output
  • look for other suspicious lesions, presence of other dysplastic naevi.

• Examination for locoregional disease:
  • examine excision site for evidence of local recurrence/residual tumour/satellite lesions
  • examine draining LN basin (may need to examine multiple areas e.g. both groins for lower mid back primary site, bilateral neck for central scalp)
  • assess for clinically palpable masses and characterise
  • if previous dissection evident, examine that area then examine for recurrence
  • comment on presence/absence of lymphoedema if previous lymph node dissection
  • complications of previous surgery; for example, nerve damage with lymph node dissection.

• Complete the systemic examination:
  • start with hands if you have time
  • examine major nodal groups (bilateral axillae/groin/cervical/supraclavicular) especially if stage III/IV disease
  • percussion and auscultation of chest to exclude pleural effusion or other features associated with lung metastases
  • palpate the abdomen for liver metastases and other intra-abdominal masses; for example, LN/retroperitoneal/bowel metastasis.

• Conclusion:
  • cover the patient
  • reposition the patient
  • thank the patient
  • clean your hands again
  • only now turn to face the examiners.
The discussion

- How do you stage melanoma?
  - Use The American Joint Committee on Cancer (AJCC) TNM system.
  - Stage I/II (Localised melanoma):
    - T1–4 N0 M0 disease
    - T stage melanoma thickness
    - Tis: melanoma in situ (tumour remains in the epidermis)
    - T1: \( \leq 1.0 \) mm thick
    - T2: 1.01 and 2.0 mm
    - T3: 2.01 and 4.0 mm
    - T4: >4.0 mm
    - each is subdivided according to absence (substage a)/presence (substage b) of ulceration
    - Breslow thickness, mitotic rate and the presence of ulceration are significant independent prognostic predictors of survival in this group of patients:
      - stage I: 85–99% 5 year survival
      - stage II: 40–85% 5 year survival.
  - Stage III (Nodal disease):
    - the number of regional LNs involved, regional node tumour burden (micro vs macro) and ulceration of the primary tumour are independent predictors of survival in this group
    - the number of involved LNs is the most important predictor of survival
    - in-transit disease (in lymphatics) and satellite lesions (of skin) are included in stage III
      - 25–60% 5-year survival.
  - Stage IV (distant metastases)
    - 15% 5-year survival.

- What is the management strategy of patients with malignant melanoma?
  - Stage I/II patients:
    - staging for metastatic disease is not indicated in asymptomatic patients due to low yield (as per NHMRC guidelines)
    - SLNB should be performed for patients with primary melanoma >1 mm in thickness. Lymphoscintigraphy with Tc99 labelled sulphur colloid and intra-operative patent blue dye
    - any palpable lymphadenopathy should be investigated with an ultrasound guided FNA of any suspicious nodes
    - all patients with stage II or more disease should be referred to a melanoma centre.
  - Stage III patients:
    - FDG PET scan with CT Brain or CT brain/chest/abdomen/pelvis to stage if the detection of occult metastatic disease would influence management
    - in-transit/locally recurrent disease should be confirmed histologically by excision/FNA biopsy of a nodule.
  - Stage IV or suspected stage IV disease:
    - FDG PET scan with CT Brain or CT brain/chest/abdo/pelvis
    - MRI brain to further characterise brain metastases may be indicated
    - tumour tissue can be tested for specific tumour mutations for consideration of targeted therapies which are available on trials for metastatic disease; for example, BRAF inhibitors for V600 mutations. BRAF and NRAS mutation testing.
What are the principles of surgical management of malignant melanoma?
Wide excision of the primary cutaneous melanoma and management of the regional lymph nodes.
The prognosis is most associated with Breslow thickness (depth measured in millimetres (mm) from the granular layer of the epidermis to the point of deepest invasion using an ocular micrometre), ulceration and mitoses.
T reatment of the primary melanoma:
- margins (NCCN guidelines):
  - melanoma in situ (Tis): 5 mm margin
  - <1 mm (T1): 1 cm margin
  - 1–2 mm (T2): 1–2 cm margin
  - >2 mm (T3,4): 2 cm margin.
  - Meta-analysis of WHO Trial, the Intergroup Melanoma Trial, the Swedish Melanoma Study Group Trial and the French Cooperative Group trial were concluded that wide (3–5 cm) margins do not improve the overall mortality compared with narrow margins in the surgical treatment of primary cutaneous melanoma (Lens et al. Excision margins for primary cutaneous melanoma: updated pooled analysis of RCTs. Arch of Surg 2007).
Management of regional lymph nodes:
- risk of LN metastases:
  - <0.75 mm: rare
  - 0.75–1 mm: 5%
  - 1–4 mm: 8–30%
  - >4 mm: 40% or higher.
- 20% of newly diagnosed stage I and II melanoma patients are considered to have an intermediate or high risk of harbouring occult regional nodal disease (elective lymph node dissection (ELND) not justified)
  - SLN identification rate with lymphoscintigraphy and blue dye is >99%
  - Breslow thickness and ulceration are the strongest independent predictors of SLN involvement
  - important prognostic indicator: 5-year survival is 56% if SLN positive vs 90% if SLN negative. Allows for staging of patient, predicts prognosis, and assists with selection for adjuvant therapies.
  - indications for SLN biopsy if clinically negative nodes:
    - Breslow >1 mm, need to discuss SLNB
    - patients with melanomas 0.75–1.2 mm with primary tumour demonstrating ulceration
    - Clark level IV or V or high mitotic rate should have discussion of SLNB (12% risk of metastases).
  - MSLT-1: Multicentre Selective Lymphadenectomy Trial 1 for primary melanomas 1.2–3.5 mm were randomised to:
    - wide excision (WE) followed by nodal observation and therapeutic TLND when developed clinical nodal disease
    - WE plus SLNB followed by complete CLND if SLN positive
    - 5-year disease free survival was 73% with WE and observation, and 78% with SLNB. Five-year survival was significantly higher in the group that underwent immediate CLND for a positive SLN compared to the group that underwent nodal observation and delayed CLND for clinical nodal recurrence (71 vs 55%). Preliminary follow up information shows a statistically significant lower
rate of distant metastasis in the SLNB group (18% vs 21%) than in the observation group. No overall survival advantage has been demonstrated yet.

- only 10–20% of patients with positive SLN are found to have additional microscopic nodal disease within non sentinel LN, so is CLND indicated? MSLT-2 is addressing this question and is currently recruiting to assess the incidence of nodal failure after removal of a positive SLND in the absence of a completion dissection, the incidence and predictors of additional positive non-SLNDs in the same basin, and the survival impact.

- Clinical nodal disease (confirmed on FNA) requires therapeutic lymph node dissection.

- Lymph node dissection for melanoma:
  - axilla: level III dissection with resection of pectoralis minor
  - groin: complete clearance of subinguinal LNs in the femoral triangle including Cloquet’s node. May be extended to an ilio-inguinal dissection of nodes in the pelvis if evidence of involvement of pelvic (obtur, internal/external iliac) nodes on imaging, gross clinical involvement of >3 groin LNs or clinically suspicious nodes high in the groin
  - neck: modified radical neck dissection, may need to add superficial parotidectomy

- patients with LN metastases need discussion in a multidisciplinary team with a view to enrolment in clinical trials

- an alternative to SLNB in older patients is to mark the SLN with lymphoscintigraphy and tattoo and undergo ultrasound follow-up. This may be appropriate as increasing morbidity of SLNB especially of the groin in older patients and even mild lymphoedema may affect the quality of life significantly.

- Treatment of locoregional recurrence:
  - persistent melanoma or melanoma with close margins should be re-excised
  - adjuvant radiotherapy should be considered for close or positive margins unsuitable for re-resection
  - in-transit metastases and satellitosis:
    - low volume disease can be re-excised
    - local treatments include radiotherapy, cryotherapy, rose Bengal injection, laser
    - rapidly progressive limb disease can be managed with isolated limb infusion (ILI), which involves percutaneous cannulation of an artery and vein, proximal occlusion of the blood supply, delivery of local chemotherapy using melphalan under hyperthermic conditions. It can achieve 90% response rates and 60–70% complete response rates. Systemic therapies and immuno modulators may be necessary.

- Regional lymph nodes:
  - SLNB should be considered if recurrence occurs and the nodal basin has not been dissected, and if no clinical evidence of nodal involvement
  - lymph node dissection for clinically involved nodes following confirmation
  - post-operative radiation therapy should be considered for adverse pathological findings
  - clinical recurrence in a previously dissected nodal basin should be managed by excision followed by RTx.

- The role of radiotherapy (RTx) in melanoma:
  - RTx to primary site for:
    - inoperable disease
• involved or close margins that cannot have further excision due to anatomical constraints
• high risk for local recurrence (e.g. Breslow >4 mm, ulceration, satellitosis, lymphovascular invasion).
• RTx to regional LN basin after LN dissection:
  • > 3 LNs involved
  • LN > 3 cm
  • extracapsular extension of clinically palpable disease
  • regionally recurrent disease.
• Palliative radiotherapy for symptomatic bone and brain metastases: brain metastases confer the worst prognosis for stage IV disease, 1 year survival 10–15%. Most patients die from complications of CNS metastases. Options for brain metastases include: surgical resection, whole brain radiotherapy (WBRT), stereotactic radiosurgery and chemotherapy. WBRT is standard treatment for melanoma brain metastases. Resection followed by WBRT is superior to WBRT alone for single metastasis.
• The role of surgery in stage IV disease:
  • staging: M1a (skin, soft tissue, LN metastases), M1b (lung metastases), M1c (visceral, brain metastases, raised LDH). Site of metastases affects prognosis
  • metastasectomy: (a) patient selection is crucial; (b) surgical resection of selected patients with metastatic melanoma in up to five sites leads to a 5-year survival of 42.5%; (c) most important prognostic factor for survival is the presence of a solitary metastasis; (d) patients with surgically operable metastasis should be considered for resection.
• Adjuvant therapies for melanoma
  • Immunotherapy:
    • high dose Interferon (IFN) alpha. Only adjuvant regimen approved by FDA for stage IIB, IIC and III patients. Improves relapse-free survival by 10% at 5 years, but no overall survival advantage. Substantial toxicity, but should be considered in patients at high risk for recurrence
    • interleukin (IL)-2. Proleukin — response in 16% of patients (not used in Australia).
  • Chemotherapy:
    • dacarbazine, temozolomide, fotemustine — complete response rates in < 5%.
  • BRAF inhibitors:
    • 60% of melanomas contain a B-Raf gene mutation. The B-Raf inhibitor, Vemurafenib, is against V600E mutation in melanoma, and causes apoptosis in melanoma cell lines which carry the mutation. On trial, used in disseminated disease.
  • Ipilimumab:
    • monoclonal antibody that binds CTLA-4 on cytotoxic T lymphocytes. Approved for use in stage IV melanoma, It causes hepatic toxicity.
• Follow-up of melanoma patients:
  • stage I: 6 monthly for 5 years then yearly thereafter
  • stage II/III/IV: 3–4 monthly for 2 years, 6 monthly until 5 years, yearly thereafter.